THE VENOM OF THE ORNAMENTED SNAKE *DENISONIA MACULATA*

by

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In 1930 Mr. Tom Eades brought back from Brigalow, in South-Western Queensland five specimens of a colour variety of this species, *D. maculata* var. devisii, Waite and Longman (1920).

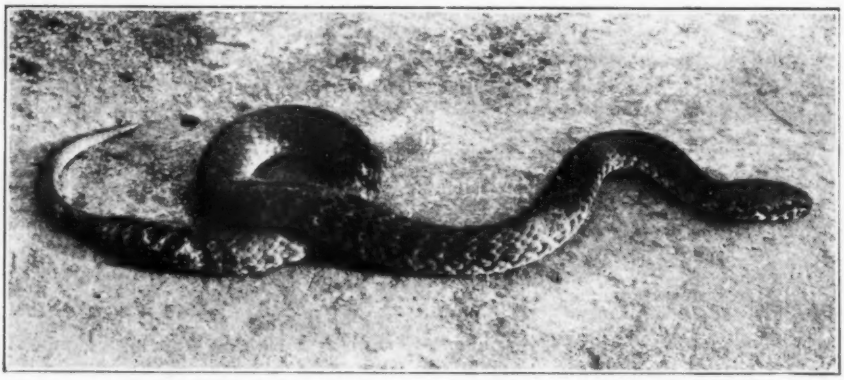


Figure 1. Photograph of *D. maculata var. devisii*, by Mr. Donald Thomson.

They were very lively and aggressive. In striking they raised the head and threw the body forward, sometimes even lifting themselves slightly off the ground. Fig. 1 is a photograph taken by my colleague, Mr. Donald Thomson, showing one of them in a characteristic attitude.

In addition to the possession of the general characters of the *Denisonias* (maxillary extending as far forward as the palatine, with anteriorly grooved fangs followed by 3 to 5 solid teeth, mandibular teeth longest anteriorly, absence of loreal, cylindrical body with scales in 15-19 rows, tail moderate or short, the subcaudals single), this species has the following special characters, illustrated by the description of one of our specimens, for which I am indebted to Mr. Donald Thomson.

Denisonia maculata Steindacher.

Locality: Brigalow, South-Western Queensland. Collected April, 1930. Length, 406·0 mm.; tail, 64·0 mm., weight, 29·0 gm.; width of head, 12·5 mm.; gape, 15·5 mm.; anal entire, subcaudals 33, abdominals 124. Transverse body scales at each 20th abdominal plate.

20th.	40th.	60th.	80th.	100th.	120th.
17	17	17	17	15	13

General Description. Head moderately distinct from body. Eye fairly large, with circular pupil and considerably longer than its distance from the mouth. Rostral small, visible from above, twice as long as broad (4·0 x 2·0 mm.). Internasal 2·0 mm., four-fifths as long as prefrontal, 2·5 mm. Frontal (5·5 x 3·5) a little more than 1½ times as broad as the supra ocular (2·5 mm.). Nasal large and entire and in contact with the single preocular. Post-oculars 2. Temporals 2 + 2, lower anterior wedged between 5th and 6th supra labials. Supra labials 6, 3rd and 4th entering eye. Three lower labials in contact with anterior chin shields.

Colour. Dull brownish, with about 60 irregular, but on close examination well-defined, transverse bands of blackish-brown which start at the nape and continue along the entire length of the body. The bands break up on the lateral line to irregular spots of reddish-brown and black. The head is uniform dark brown, the sides of head, supra and infra labials variegated with brown and yellowish.

This snake grows to a length of 400 mm. (about 16 inches) or more, and according to Kinghorn (1929) is found in the northern half of New South Wales and Southern Queensland. The three largest of our specimens were 406, 370, and 336·5 mm. long.

The combined venom yields of the two largest on February 20 and March 19, 1930, were 2·4 and 3·0 mg., and on May 13 four snakes yielded 4·2 mg. of dry venom. The largest individual yield was 1·9 mg. of dry venom.

The toxic action of this venom was studied by intravenous injection in rabbits and subcutaneous injection in guinea-pigs.

Effects of Intravenous Injection in Rabbits.

The venom was injected intravenously into a marginal ear vein of domestic rabbits in a concentration of 1 mg. per c.c. The results are set out in Table 1.

TABLE 1.

The Results of the Intravenous Injection of the Venom of D. maculata in Rabbits.

Number of Animals.	Weight or average weight in kilos.	Dose in mg. per kilo.	Result.
1	1·04	1·0	Died in 13 minutes.
5	2·11	0·4	Three died in 21, 22, and 26 minutes, 1 in 2½ hours, and 1 survived.
1	0·91	0·35	Collapsed in half an hour, and remained so, with occasional convulsions, for 2½ hours. Dramatic recovery.
1	1·39	0·3	Collapsed in 1 hour, and remained so, with occasional convulsions for 2 hours. Suddenly recovered.

The certainly lethal dose for rabbits is rather more than 0·4 mg. per kilo, and the venom, like that of the copperhead, is devoid of thrombin.

Symptoms.

The onset is marked by a loss of tone more sudden than in poisoning by copperhead venom. The head falls over on one side, and shortly the whole body becomes slack. The pupils are contracted; the corneal reflex is lost a few minutes before death, which occurs by respiratory failure. The symptoms suggest a predominantly "neurotoxic" action.

Post-mortem.

The blood was fluid and the heart was still beating immediately after death; the clotting time was delayed (45 minutes, 1 hour, and rather more than one hour in three observations). There was no obvious haemolysis nor any lesions in the organs, probably because the time to death was so short.

A few experiments were made in which venom was injected into rabbits under anaesthesia with the blood pressure recording to ascertain whether the sudden loss of tone and dramatic recovery in animals receiving a sublethal dose of venom intravenously were due to changes in blood pressure. The injection of this venom produces no immediate effect upon the blood pressure of the anaesthetized rabbit. If the dose is a lethal one the blood pressure falls gradually. It does not reach a low level, but commences to rise when the respiration changes in type, becoming first dyspnoeic as the accessory muscles of respiration are called into play and then wholly costal. At this stage the blood is insufficiently oxygenated and the blood pressure is high, and shows Traube Hering waves. Faradic stimulation of the phrenic roots in the neck fails to cause contraction of the diaphragm. Shortly after this, respiration fails and there are general convulsions. If artificial respiration be not applied, the animal dies, but with adequate artificial ventilation it may survive some hours, and, in the case of this venom, the peripheral paralysing effect on the phrenic motor endings may pass off and the animal again breathe naturally. The following experiment gives the details of one such observation.

Experiment 1 (Fig. 2) Domestic rabbit, 1·75 kilos.

10.50 a.m. 0·8 c.c. of 10 p.c. "Numal" intravenously.

11–11.15 a.m. Under additional light ether anaesthesia a tracheal cannula was inserted and the upper root of the right phrenic nerve was exposed in the neck. Faradic stimulation with the coils at 50 cm. separation gave powerful contractions of the diaphragm, visible and readily palpable at the costal margin. Rectal temperature 38·3°C.

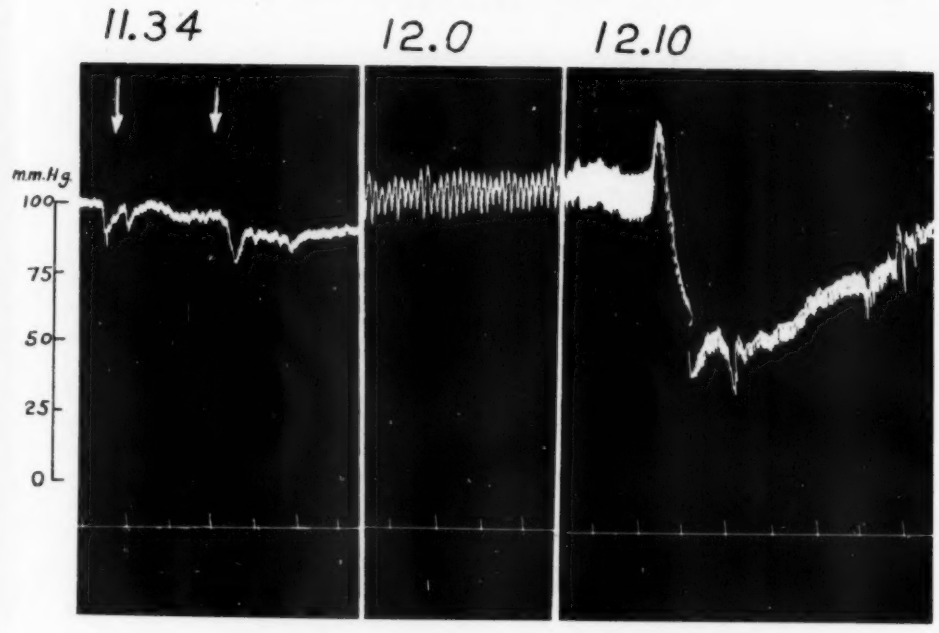
11.17 a.m. Blood pressure recording 104 mm. Hg., respiration 47 per minute.

11.34–11.35 a.m. 1·2 mg. of the venom of *D. maculata* in a concentration of 1·0 mg. per c.c. injected into a marginal ear vein.

11.36 a.m. Blood pressure 90 mm. Hg.
11.37 a.m. Blood pressure 86 mm. Hg. Respiration 47 per minute.
11.49 a.m. Dyspnoea—change in type of respiration, 56 per minute.
11.54 a.m. Blood pressure 100 mm. Hg.
12.0 noon. Strong dyspnoea—respiration 46 per minute. Mean blood pressure 104 mm. Hg. Traube Hering waves.
12.5 p.m. Respiration 37 per minute, wholly costal, no response to stimulation of the right phrenic root at 14 cm. separation of the coils. Blood dark and asphyxial.
12.6 p.m. Strong vagal effect apparent in blood pressure tracing. Respirations 35 per minute.
12.11 p.m. Respiration ceased. Artificial respiration commenced—immediate fall of blood pressure to 42 mm. Hg.
12.15 p.m. Faradic stimulation of the right phrenic nerve with the coils separated 5 cm. No response. Blood pressure 100 mm. Hg.
12.45 p.m. Blood pressure 72 mm. Hg. Artificial respiration stopped for over a minute—contractions of alae nasi commenced at once, but no effective respirations—immediate asphyxial rise of blood pressure. No response of the diaphragm to stimulation of the phrenic nerve.
3.16 p.m. Artificial respiration again discontinued, good respiratory response but blood in cannula clotted.
4.20 p.m. Other carotid cannulized. Blood pressure 118 mm. Hg.
4.30 p.m. Blood pressure 108 mm. Hg. Artificial respiration discontinued. Respiration 120 per minute, adequate and diaphragmatic. Good response to Faradic stimulation of the phrenic nerve with the coils separated 50 cm.
4.36 p.m. Blood pressure 98 mm. Hg. Respiration 112 per minute.
4.45 p.m. Blood pressure 78 mm. Hg. Respiration 100 per minute.
5.20 p.m. Blood pressure 78 mm. Hg. Respiration 100 per minute.
6.3 p.m. Blood pressure 73 mm. Hg. Animal in good condition, coming out from anaesthetic. The experiment was discontinued and the animal killed.

This experiment is noteworthy because with no other venom have we observed a paralysis of the phrenic endings which recovered spontaneously without neutralization *in vivo* by antivenene. It does not, however, throw much light on the curious behaviour of rabbits injected with sublethal doses of venom unless the sudden loss of tone is a spontaneously reversible peripheral effect (masked in these experiments by anaesthesia) and other motor nerve endings are more sensitive than those of the muscles of respiration. The fall of blood pressure following the injection of venom is too small to account for the typical collapsed and toneless condition seen in the non-anaesthetized animal.

Figure 2. Blood pressure tracing from a domestic rabbit injected intravenously with 1·2 mg. of the venom of *D. maculata*. The tracing reads from left to right and the time is in half-minutes. Between the arrow heads in the first piece of tracing, starting at 11.34 a.m., the venom was injected. The second, at 12.0 noon, shows Traube Hering curves. The third, at 12.10 p.m., shows the effect of cessation of breathing—strong vagal slowing of the heart and a sudden fall and gradual recovery of blood pressure following artificial ventilation. The fourth, at 12.45 p.m., shows asphyxia following temporary cessation of artificial ventilation for a little over a minute. The fifth, at 4.30 p.m. shows the effect of permanent cessation of artificial ventilation. The last tracing is at 6.0 p.m.


11.34
12.0
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mm.Hg.
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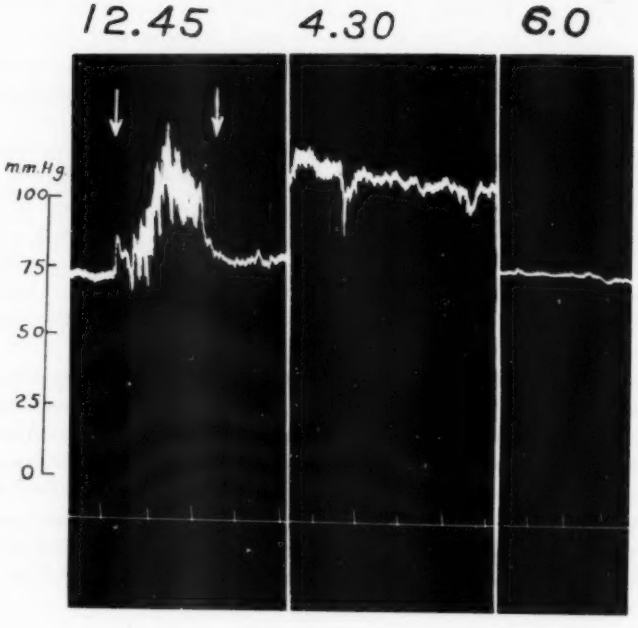
12.45
4.30
6.0
mm.Hg.
100
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25
0

Effects of Subcutaneous Injection in Guinea-pigs.

The venom was injected subcutaneously into the tissues of the abdominal wall in a concentration of 1 mg. per c.c. The results are set out in Table 2.

TABLE 2.

Results of Subcutaneous Injection of the Venom of D. maculata in Guinea-pigs.

Number of Animals	Weight or average weight in grams.	Dose in per 100 gm.	Result.
1	200	0·25	Died in 52 minutes.
2	208	0·2	Died in 49 and 54 minutes.
4	230	0·15	Died in 43, 52, 54, and 60 minutes.
2	215	0·12	Died in 55 minutes and in 1 hour 52 minutes.
2	219	0·1	Died in 1 hour 20 minutes and 1 hour 39 minutes.
2	207	0·08	Died in 1 hour 15 minutes and in 1 hour 17 minutes.
2	220	0·06	Died in 2 hours 38 minutes and in 5½ hours.
6	218	0·05	1 died in 7 hours and 5 survived.
2	212	0·04	Both survived without symptoms.
2	211	0·02	Both survived without symptoms.

The certainly lethal dose for guinea-pigs is probably about 0·6 mg. per kilo.

The symptoms were strikingly like those of bronchial obstruction, the animals appearing to have great difficulty in getting air into their lungs. In addition there was collapse and, in some, obvious paralysis of the hind limbs, and "starting movements" occurred from time to time. In many the nose and mouth were wet, either from salivation or from excessive secretion from the respiratory tract. Death occurred from respiratory failure, and the heart was still beating and the blood dark when autopsy was made immediately after death. In a few animals the lungs were found distended, filling the thoracic cavity and overlapping the heart, but in most they were neither collapsed nor distended, but in the mid-way position when the chest was opened. The lungs were congested and petechial or more extensive haemorrhages were observed. There was only slight oedema at the site of injection. The clotting time of the blood at room temperature, as observed in a clean tube 15 mm. internal diameter, was only a little delayed—the times in three observations being 5, 7, and 16 minutes. In one or two animals the adrenals were congested.

The venom is not so toxic as that of the copperhead *D. superba*, although the intravenous lethal dose for the rabbit is the same for both venoms—the sudden and complete recovery of animals after the production of severe symptoms with an intravenous dose which is just sublethal is not paralleled by the behaviour of rabbits injected with sublethal doses of copperhead venom. A further difference between the venoms is displayed by the results of subcutaneous injection in guinea-pigs for which the certainly lethal dose for *D. superba* venom is 0·06 mg. per kilo, and for this venom about ten times as great. With this venom if death

does not occur within 7 hours recovery is likely. There were no late deaths on the 2nd, 3rd, or 4th day, as in guinea-pigs injected subcutaneously with the venom of *D. superba*.

The haemolytic power of the venom was tested by Miss Williams upon 3 p.c. washed horse, rabbit, and guinea-pig corpuscles, and upon defibrinated blood diluted to an equivalent extent. The haemolytic system had a volume of 0·3 c.c. (0·1 corpuscles 0·2 saline containing various doses of venom (0·1 to 0·1 x 10^{-6} mg.)). Washed guinea-pig corpuscles showed a trace of haemolysis in 20 hours with 0·1 mg. of venom and diluted defibrinated horse blood showed showed rather more haemolysis with 0·1 and 0·01 mg. of venom. Otherwise no haemolysis was observed.

These results indicate how much less potent as a haemolytic agent this venom is than the venom of the copperhead, *D. superba*, which causes complete haemolysis with washed horse and rabbit blood cells in 1½ hours at a dose of 0·1 mg., and in 3 to 5 hours at a dose of 0·01 mg.

In none of the experimental animals was there intravascular haemolysis or haemoglobinuria.

The species is described by Kinghorn as venomous but not dangerous. The experience of our collector, Mr. Eades, in Chinchilla in February, 1930, inclines me to modify this opinion. Mr. Eades was packing up his snakes preparatory to returning to Melbourne, and, in transferring some specimens of *D. maculata* from a tin, the largest fastened on his finger. He disregarded the bite and did not treat it, but went on with his work. Half an hour later, while on his way to the house of his host, a distance of less than 100 yards, he was overcome by the venom, "knocked out as by a blow", and fell down unconscious. He recovered consciousness just as suddenly 12 hours later in the Chinchilla Hospital, where he was under the care of Dr. Hill.

Though it is possible that anaphylactic shock may have played a part in this picture, since Eades had been bitten many times by snakes—once or twice by the copperhead, *D. superba*—and is very sensitive to snake venom, it is at least interesting that sudden loss of tone and sudden recovery were observed following the intravenous injection of the venom in sublethal doses into rabbits.

CONCLUSIONS.

The venom of this small species of *Denisonia* is rather less potent than that of the copperhead, both in respect of its haemolytic and "neurotoxic" properties. The venom has a strong paralysing action on the phrenic motor endings in the diaphragm which is spontaneously reversible. A bite in man was followed after an interval by sudden loss of consciousness, from which recovery occurred with dramatic suddenness some hours later. A somewhat similar phenomenon was observed in rabbits injected intravenously with sublethal doses of venom.

ACKNOWLEDGMENT.

My thanks are particularly due to my one-time colleague, Mr. Donald Thomson, for the photograph of fig. 1, and the description of one of our specimens, and to Miss F. E. Williams for the observations on haemolysis.

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HAEMOLYSIS BY AUSTRALIAN SNAKE VENOMS

3. SOME FACTORS WHICH INFLUENCE THE ACTION OF THE VENOM OF THE COPPERHEAD

by

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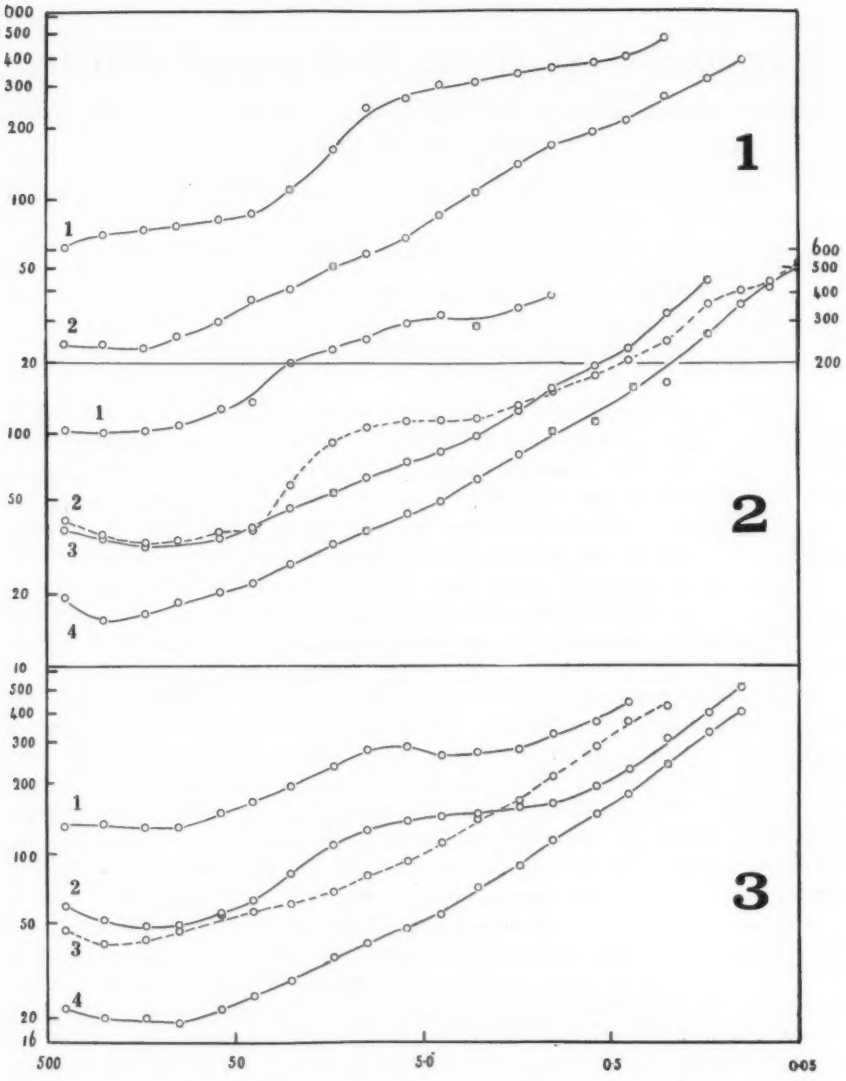
(Submitted for publication 2nd May, 1934.)

An extensive survey was made of the haemolytic activities of the venoms of Australian snakes by Kellaway and Williams (1933). They investigated, among other matters, the effect of adding lecithin, the inhibition by sera, and the changes due to the absence of ions from the fluid in which the venoms were dissolved and the cells suspended. The present paper deals only with the influence of a limited number of factors on the haemolysis of rabbit erythrocytes by the venom of a single species of snake, the copperhead. The effects studied were those due to variations of the hydrogen ion concentration, variations in the concentration of the red cells, the presence of a tri-valent anion, and the addition of certain proteins.

The method employed was that due to Ponder (1921), modified owing to the longer time required for haemolysis. 10–12 c.c. of rabbit blood was freshly drawn into 15 c.c. of 2 p.c. sodium citrate solution in a 90 c.c. centrifuge cup. The cup was filled with the solution with which the cells were to be washed and the mixture centrifuged. After withdrawal of the supernatant liquid the cells were washed four times with 80 c.c. of the same solution. The venom and proteins were dissolved in a solution of the same composition as that with which the cells were washed, and with which all dilutions were made.

Mixtures of solutions containing the venom, and proteins when used, to a total volume of 1·0 c.c., were placed in small tubes and placed in a water-bath at 37·3°C. 0·4 c.c. of a 5 p.c., by volume, suspension of washed rabbit cells was added, the contents mixed, and the time noted. A series of some 40–50 tubes was thus put up in a few minutes, without much change of temperature, and with the cells meeting the venom at substantially the same time after removal from the rabbit. For the initial experiments 0·9 p.c. solution of sodium chloride was used for washing the cells and dissolving the venom and proteins. It was found, however, that this is not a suitable medium for the purpose. Although on any given day a regular set of curves could be obtained, the results from day to day varied con-



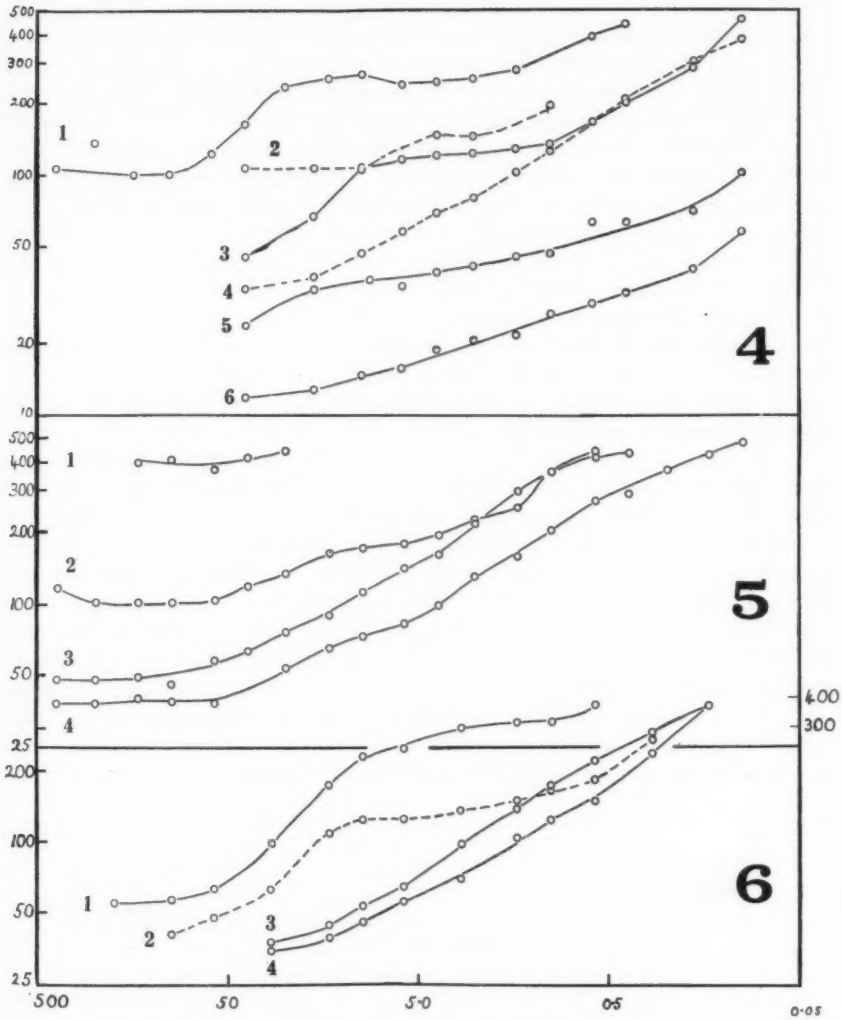
Abscissae: milligrams of copperhead venom per c.c. of cells.
Ordinates: time for complete haemolysis in minutes.
Both are plotted on a logarithmic scale.

siderably, particularly when different rabbits were used as the source of cells. It would appear that the concentration of hydrogen ions has a great influence on the course of haemolysis, and that to ensure stability in this matter recourse must be had to a buffer solution both for washing the cells and for the subsequent experiments. The intact cell under the conditions of these experiments does not appear to control the reaction of the fluid in which it is suspended. Some experiments were performed using mixtures of phosphates, but, as shown by Bang (1909), this ion delays haemolysis. Although, over the range studied, acetate buffers have very little reserve acidity, their reserve alkalinity is ample for this purpose, and they may be used in relatively high concentration without seriously extending the time of haemolysis. Accordingly, unless otherwise mentioned, the solution used contained 8·10 gm. of sodium chloride and 1·19 gm. anhydrous sodium acetate per litre, adjusted with acetic acid to the requisite hydrogen ion concentration. The effect of replacing the acetate ion by phosphate at the same hydrogen ion concentration and of variations in the acidity may be seen in fig. 5. As is shown in figs. 1 and 6, the substitution of sodium acetate for one-tenth of the sodium chloride appears to have no effect.

Variation of the concentration of hydrogen ions not only alters the time of haemolysis, but modifies considerably the form of the time-dilution curve. At pH 5·3–6·0 the curve approximates empirically to log. time $\propto$ log. concentration of venom for most of the time during which observations were made. At high concentrations of venom there is the inhibition described by Kellaway and Williams (1933), and at great dilutions the destruction of the venom due to the acid solution deviates the curve towards the axis of time. At pH 6·6 the curve, in addition to its initial inhibition, has a portion flattened with respect to the axis of concentration, within which a dilution eight- to ten-fold produces little change in the time of haemolysis. For more alkaline solutions the curve has a slightly different form again, the flattened portion being shorter, but preceded by a longer portion concave towards the axis of concentration of venom.

Figure.	Curve.	
1	1	0·9 p.c. NaCl containing 0·013 p.c. $NaHCO_3$. pH 8·0.
	2	0·9 p.c. NaCl containing CO_2. pH 5·6.
2	1	in 0·08 p.c. denatured ox serum albumin. pH 6·6.
	2	control for 1.
	3	in 0·08 p.c. denatured ox serum albumin. pH 6·0.
	4	control for 3.
3	1	in 0·02 p.c. native ox serum albumin. pH 6·6.
	2	control for 1.
	3	in 0·02 p.c. native ox serum albumin. pH 6·0.
	4	control for 3.

The curves in figures 2 and 3 represent haemolyses in the standard acetate-sodium chloride buffer solution.



Abscissae: milligrams of copperhead venom per c.c. of cells.
Ordinates: time for complete haemolysis in minutes.
Both are plotted on a logarithmic scale.

Variation of the concentration of red cells from 6·6 p.c. by volume to 1·3 p.c. had no effect on the time-dilution curve, provided that the concentration of venom was varied concomitantly.

The effect on the haemolysis of red blood cells by copperhead venom of five proteins was studied: native and denatured ox serum albumin, casein, crystalline hen ovalbumin, and rabbit haemoglobin. The serum albumin was denatured by heating for several hours at 80°C. in 0·5 N. HCl, precipitating and washing several times at its iso-electric point and drying with alcohol and ether. The haemoglobin was added in the form of red cells laked with water; the solution was adjusted to the standard salt concentration before use. The solutions of the proteins were adjusted before use to the same concentrations of hydrogen and of other ions as the buffer solutions in the particular experiment.

The haemoglobin had a strongly accelerative effect on the course of haemolysis; the effect was greatest at pH 6·0, and least at pH 8·0 (see fig. 4). The other proteins were all inhibitory, decreasing in effect in the order named. Their inhibitory powers were less at pH 6·6 than at pH 6·0. (See figs. 2, 3, 7, 8.) The difference between serum albumin and egg albumin is notable, the latter having very little effect, particularly at pH 6·0, where over much of the curve it is faintly accelerative.

Whereas casein tended to smooth out the flat portion of the curve, the serum albumin and its denatured derivative increased the discontinuity in the curve for pH 6·6. The existence of a *maximum* in this region has been observed so many times that there seems no ground for doubting it. It is, in general, enhanced by the presence of serum albumin.

If, from the curves of inhibition for pH 6·0, the amounts of venom rendered ineffective by the protein be calculated the result would suggest some adsorption compound, analogous to that found by Ponder (1925) in the case of serum protein

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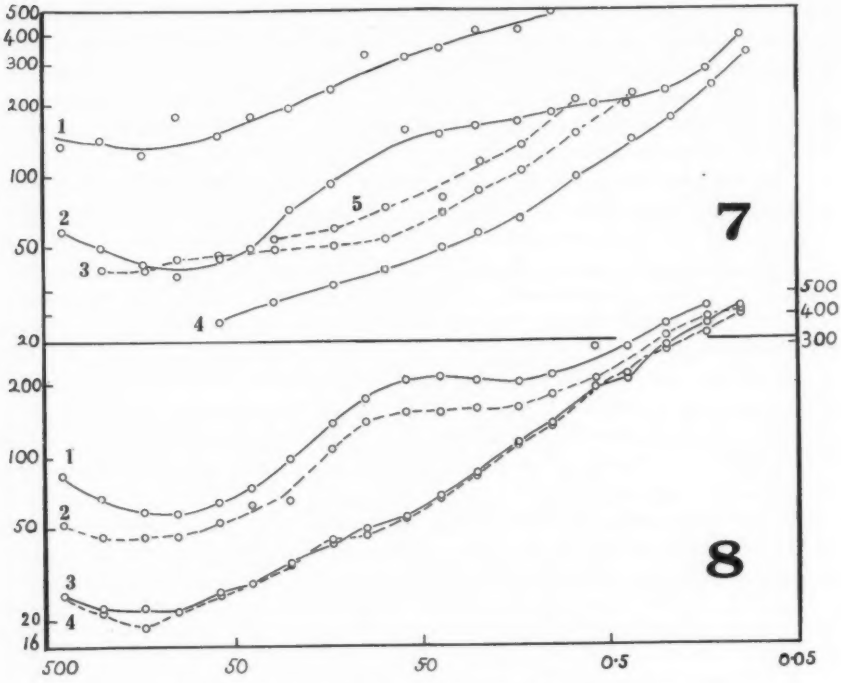
Figure.	Curve.	
4	1	control for No. 2.
	2	in 2 p.c. laked cells in solution for curve 1, figure 1.
	3	control for No. 5.
	4	control for No. 6.
	5	in 2 p.c. laked cells. pH 6·6.
	6	in 2 p.c. laked cells. pH 6·0.
5	1	in 0·02 M phosphate buffer in 0·81 p.c. NaCl. pH 7·6.
	2	in 0·02 M phosphate buffer in 0·81 p.c. NaCl. pH 6·6.
	3	in 0·02 M phosphate buffer in 0·81 p.c. NaCl. pH 5·6.
	4	in 0·02 M acetate buffer in 0·81 p.c. NaCl. pH 5·6.
6	1	acetate buffer in 0·81 p.c. NaCl. pH 7·2.
	2	acetate buffer in 0·81 p.c. NaCl. pH 6·6.
	3	acetate buffer in 0·81 p.c. NaCl. pH 5·6.
	4	acetate buffer in 0·81 p.c. NaCl. pH 6·0.

The curves 3–6 in figure 4 represent haemolyses in the standard acetate-sodium chloride buffer solution.

and saponin. While such may well be for saponin, the improbability of such a compound with venom has been shown by Kellaway and Williams (1933) in their experiments on prehaemolytic swelling of erythrocytes. An experiment was carried out in which a solution containing casein and copperhead venom, in



Abscissae: milligrams of copperhead venom per c.c. of cells.
Ordinates: time for complete haemolysis in minutes.
Both are plotted on a logarithmic scale.

Figure. Curve.

7
1 in 0·8 p.c. casein. pH 6·6.
2 control for No. 1.
3 in 0·4 p.c. casein. pH 6·0.
4 control for Nos. 3 and 5.
5 in 0·8 p.c. casein. pH 6·0.

8
1 in 0·8 p.c. crystalline ovalbumin. pH 6·6.
2 control for No. 1.
3 control for No. 4.
4 in 0·8 p.c. crystalline ovalbumin. pH 6·0.

The curves in figures 7 and 8 represent haemolyses in the standard acetate-sodium chloride buffer solution.

amounts which showed an effective inhibition, was adjusted to pH 4·6 and centrifuged. The supernatant fluid contained the whole of the haemolysin of the venom, and gave the same time-dilution curve as the control solution. The difference between copperhead venom and saponin is clearly shown in the acceleration of the former and inhibition of the latter by haemoglobin. It is of interest to note that ox serum albumin inhibits both strongly, while egg albumin has relatively little effect on either. Another similarity may also be noted. If some red cells be laked with water and made up to a 5 p.c. suspension in 0·9 p.c. sodium chloride solution, the solution is turbid, owing to the presence of suspended stroma protein. If a trace either of saponin or of copperhead venom be added the protein is dissolved and the solution becomes quite clear.

SUMMARY.

The hydrogen ion concentration affects the velocity of haemolysis of rabbit cells by copperhead venom. The inhibitory effect of proteins on this reaction was studied. Haemoglobin accelerates the haemolysis.

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ENERGY METABOLISM AND INSENSIBLE PERSPIRATION OF AUSTRALIAN ABORIGINES

by

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(Submitted for publication 12th March, 1934.)

The aborigines of Central Australia live under conditions of high temperature and low humidity during a considerable part of the year. The periods during which they are most likely to suffer from lack of water are those in which from physiological considerations they should need more of it. They must often lose, not only the whole of their heat production by evaporation, but also what they absorb, owing to high external temperatures. They should need more water than persons in temperate, well-watered regions, unless they have developed some means of sparing it. They are indeed able under certain conditions to reduce both their voluntary activity and their basal heat production very materially (Wardlaw and Horsley, 1928), but in their natural state it cannot be possible for them to lead a very slothful existence. These two means of sparing heat production are not independent, as several investigators have shown (Benedict and Smith, 1915; Ozorio de Almeida, 1920; Wishart, 1927). This is very evident in Australian aborigines (Wardlaw and Lawrence, 1932).

In the present investigation the measurement of the proportion of the basal heat lost from the body by evaporation has been the principal object. A few measurements have been made under conditions other than basal and over periods up to twenty-four hours.

The Aboriginal Subjects.

Tribe: With the exception of Nos. 3, 4, and 14, who were full-blood Aranda, the subjects were full-blood Aluritja.

Age and Sex: Adult males.

Activity: No regular physical activity was allowed during the period of observation. Up to that time the Aluritja had been living under natural conditions. The Aranda had been employed as stockmen.

Diet: This consisted of 450 gm. flour, 80 gm. sugar, and a little tea as rations, supplemented to an unknown extent by rabbits; no hunting was engaged in, nor was any attempt made to collect native vegetable foods.

Health: With the exception of No. 15 (who showed signs of nephritis) and No. 22 (who had been treated for yaws), the subjects were in normal health and had not recently suffered from any illness.

Climatic Conditions: These are summarized in Table 1. In brief, the region was hotter, drier, and higher than those in which the previous observations were made. The small difference between the average temperatures at which the basal observations were made in Sydney and Hermannsburg is due to the fact that the Sydney observations were made much later in the morning.

TABLE 1.

Meteorological and Geographical Data.

Place.	Lismore (Kyogle)	Walgett (Angledool)	Alice Springs (Hermannsburg)	Sydney.
Latitude	28°48′ S	29°6′ S	23°57′ S	33° 52′ S
Longitude	145°0′ E	147°54′ E	132° 45′ E	151°12′ E
Miles from coast	35	270	570	4
Feet above sea level	150	450	1,640	138
Mean annual temperature	19·3°C	19·9°C	21·1°C	17·3°C
Mean annual relative humidity	76%	51%	38%	70%
Mean annual rainfall (inches)	35·0	18·5	10·8	47·5
Mean temperature of experiments	25·3°C	13·5°C	25·6°C	24·2°C
Mean relative humidity of experiments	80%	54%	44%	53%
Period of observations	Jan.–Feb. 1928.	Aug.–Sept. 1929.	Jan.–Feb. 1933.	Oct.–Nov. 1932.
Mean maximum temperature for months	29·4°C	19·0°C	36·1°C	22·7°C
Mean relative humidity for months	75%	54%	30%	69%

The White Subjects.

The same measurements were made on these as on the aborigines. Their activity and diet in Central Australia were not significantly different from what they were in Sydney.

Physique and State of Nutrition.

The heights and weights of the nude subjects are recorded to 0·5 cm. and 0·2 kg. The weights are also expressed as percentages of the weights for given heights and ages reported by Hunter (1912). The ages had to be assessed. The effect of age on weight is more important in the younger subjects, but it is not likely that the assessed ages were outside of the correct five-year age groups of the tables used.

The sitting heights (knees drawn up) were also recorded to 0·5 cm. and the pelidisi calculated.

Basal Metabolism.

The Haldane-Douglas method was used under conditions similar to those of previous investigations (Wardlaw and others, *loc. cit.*).

Although the subjects were co-operative, many repetitions were necessary

with some before evidence of steadiness was obtained. Even then some R.Q.'s showed evidence of over-breathing. The values must therefore be regarded as maximal. Results differing by more than 5 p.c. from the mean were rejected.

Insensible Perspiration.

For basal measurements the subjects were weighed before and after about 30-minute periods (Benedict and Root, 1926) on an Oertling balance sensitive to 0·1 gm. The maximal error is 3 p.c. of the change of weight in this period. The subjects lay on a steel mattress. Textile material was limited, on account of its hygroscopic properties (Heller, 1930), to a duck cover for the mattress, two air cushions, and, for nude subjects, a blanket. Clothed subjects wore shirt and trousers only.

Net extrarenal water loss was calculated by deduction of the difference between weights of CO_2 lost and O_2 absorbed during the period. This correction is zero at R.Q. = 0·73, but reaches 25 p.c. at R.Q. = 1·0. Loss of CO_2 through the skin has been neglected. According to Barratt (1897) and Shaw and Messer (1930) this never reaches 2 p.c. of the total loss, and is usually much less.

Relative water losses from the skin and respiratory passages have been calculated on the assumption that expired air contains 34 mg. water per litre (Osborne, 1910; Galleotti, 1912; Liljestrand and Sahlstedt, 1925; von Czike, 1931). Only slight variations from this figure are shown by the different investigators, except under extremes of temperature and humidity (Osborne, 1913; Galleotti, 1915) and certain abnormal conditions (von Czike, 1933). Jacquot and Mayer (1925) and Christie and Loomis considered that variations of humidity of expired air are explained by variations of body temperature.

The Smithsonian tables were used in the calculations, which were further checked by a nomogram constructed by W. H. H. Gibson, B.Sc., B.E., from tables calculated by C. E. Corlette, M.D. (1931) (personal communication).

RESULTS AND DISCUSSION.

Physical measurements.

These are given in Table 2. The average height of the subjects is 0·7 cm. less than that of the two groups previously examined in New South Wales. It is about the same amount greater than that of the South Australian aborigines of Hicks, Matters, and Mitchell (1931).

The average weight of the subjects (54·7 kg.) is markedly lower than of the previous groups (63·0 and 69·3 kg.). A lower plane of nutrition is also indicated by their relative weight (88·4 p.c.) and pelidisi (98·6), the values for the other groups being 92 p.c. and 110 p.c. and 101 and 107 respectively.

TABLE 2.

Summary of Physical Measurements.

Subject.	Age.	Height.	Sitting height.	Weight.	Standard weight.	Surface area.	Weight obs. / Standard wt.	Pelidisi.	Basal metabolism Aub-Du Bois.	Basal metabolism Harris-Benedict.	Mouth temperature.	p.c. Calories evaporated.
Male White Subjects.												
(1) *In Sydney, N.S.W.*												
16	43	169·1	86·6	63·4	65·0	1·73	97·6	97·7	94·7	100·7		28·7
17	20	167·6	86·9	57·1	59·0	1·63	97·0	87·9	87·0	86·6		28·4
18	21	177·3	93·5	72·5	65·0	1·90	111·6	88·7	77·7	77·4		45·0
19	38	180·4	95·9	88·9	75·0	2·09	118·5	99·9	100·3	102·2		31·9
				70·5		1·84	106·2	98·8	94·0	96·6		29·7
(2) *In Hermannsburg, C.A.*												
16	43	169·1	86·6	61·2	65·0	1·69	94·2	94·0	96·3	102·4	36·9	25·6
17	20	167·6	86·9	54·4	59·0	1·60	92·3	82·8	87·8	88·4		39·8
18	21	177·3	93·5	69·4	65·0	1·86	106·7	85·0	94·6	93·8	36·7	40·0
19	38	180·4	95·9	82·9	75·0	2·02	110·6	94·1	90·7	90·5	36·2	23·5
Mean		173·6		67·0		1·79	100·9	91·0	92·4	93·8		32·2
Male Central Australian Aborigines.												
4	40	165·5	83·0	55·8	61·0	1·61	91·5	97·5	61·6	66·1	35·8	38·4
5	30	177·0	86·5	57·2	72·0	1·71	79·0	86·1	98·4	104·2	36·1	39·9
6	45	165·4	79·8	51·7	63·0	1·56	82·1	102·0	67·0	74·8	35·8	33·3
8	18	167·0	79·0	58·5	55·0	1·65	106·5	118·5	84·7		36·3	45·7
9	25	170·0	83·0	53·5	60·0	1·61	89·3	93·7	77·6	79·7	35·4	38·8
10	20	169·0	82·0	53·1	60·0	1·60	88·5	96·1	97·1	100·1	36·1	40·8
12	35	168·0	84·5	57·6	62·0	1·64	93·0	95·7	96·1	101·7	35·5	42·4
13	65	175·5		57·1	72·0	1·69	79·3		83·5	95·5	35·4	34·6
14	40	168·5	79·5	50·8	63·0	1·57	80·7	101·0	69·2	76·0	35·6	39·2
15	17	167·8	81·0	51·7	55·0	1·57	94·1	97·5	87·1		35·9	56·0
Mean		169·3		54·7		1·62	88·4	98·6	82·2	87·3	35·8	40·9

Up to the time of examination the present group had been living under natural conditions of diet and activity. The other groups had been living on European food for a period of years, many of them under conditions involving very little physical exertion. The indications are not that the present subjects were under-nourished, but that some of the previous groups were abnormally heavy.

Basal Metabolism.

Average figures for this are given in Table 2 and individual results in Table 3. The group average is 82·8 p.c. (Aub-Du Bois, 1917), 5 p.c. below the active New South Wales group (Wardlaw and Lawrence, 1913), and the overfed South Australian group (Hicks, Matters, and Mitchell, 1931). Some low values occur, but none as low as some in the inactive New South Wales group (Wardlaw and Horsley, 1928).

TABLE 3.

Basal Metabolism—Individual Results.

Subject.	Lung vent. l./min.	CO_2% prod.	O_2% abs.	R.Q.	Cals. m²/hr.	p.c. of Standard A–DB	p.c. of Standard H–B	Pulse.	Resp.
Whites, Sydney.									
16	3·953	4·23	5·58	0·758	36·4	94·6	100·2	58	6
	3·898	4·24	5·70	0·745	36·5	94·9	101·1	59	8
	3·955	4·34	5·85	0·742	38·0	98·1	105·2	56	9
	3·742	4·47	6·32	0·706	38·4	99·8	106·5	55	9
	3·350	4·43	6·08	0·729	33·3	86·5	92·6	56	6
	3·378	4·53	6·18	0·733	34·2	88·8	94·9	57	6
	3·765	4·58	5·81	0·788	36·2	94·1	100·5	52	7
17	3·375	4·67	6·19	0·755	36·5	92·4	92·1	57	8
	3·151	4·68	5·80	0·807	32·4	82·0	81·7	55	8
18	4·400	3·91	4·82	0·792	32·1	82·1	81·7	54	4
	4·690	3·87	4·95	0·761	34·8	88·2	87·6	58	16
	3·735	3·94	4·93	0·800	27·9	70·7	70·3	58	13
	3·850	3·94	4·73	0·833	27·8	70·4	70·0	58	14
19	5·992	3·49	4·81	0·726	39·0	99·8	101·6	60	9
	5·432	3·97	5·26	0·755	38·8	99·5	101·3	60	9
	5·719	4·00	5·25	0·762	40·9	104·9	106·8	64	8
	5·225	3·92	5·34	0·734	37·8	96·9	98·7	64	9
Whites, Central Australia.									
16	3·779	4·95	5·76	0·859	37·7	98·7	104·9	61	13
	3·779	4·36	5·65	0·771	36·1	93·8	100·0	62	10
	3·624	4·84	6·42	0·754	39·2	101·8	108·3	60	9
	3·486	4·54	5·83	0·779	34·4	89·4	95·2	58	12
17	2·792	5·14	6·53	0·809	32·9	83·3	84·0	48	5
	2·606	4·79	6·10	0·785	28·5	72·2	72·8	49	7
	3·004	4·96	6·54	0·758	35·0	88·7	89·3	52	6
	3·242	4·94	6·06	0·815	35·6	90·1	90·8	52	6
18	4·081	4·87	6·13	0·794	38·7	97·7	97·6	60	12
	4·079	4·30	5·72	0·752	35·7	90·4	90·1	60	11
19	5·026	3·92	5·29	0·741	37·3	95·6	98·2	55	18
	5·063	3·88	5·03	0·772	36·0	92·3	95·0	55	18
	4·743	3·94	5·59	0·704	37·0	94·9	97·2	62	16
	5·109	3·83	5·09	0·752	36·6	93·9	96·6	62	14
	4·709	3·92	4·86	0·806	32·7	85·0	87·4	60	16
	4·831	3·87	5·12	0·755	34·9	89·5	92·1	56	18
Aborigines, Central Australia.									
4	2·741	3·56	5·05	0·703	24·2	62·9	67·4	58	16
	2·774	3·74	4·68	0·800	23·2	60·2	64·8	58	12
45	4·986	4·10	4·53	0·905	39·1	98·7	104·6	60	18
	4·887	4·09	4·60	0·890	38·7	98·0	103·7	63	16
6	3·390	3·52	4·08	0·862	25·9	67·2	74·3	50	14
	3·739	3·54	3·59	0·986	26·0	67·2	74·3	52	13
8	5·114	3·72	3·66	1·017	34·3	83·7	86·4	68	14
	5·327	3·63	3·60	1·010	35·2	85·6	88·4	60	17
9	4·338	3·71	3·91	0·948	31·5	79·8	82·0	49	19
	4·527	3·49	3·50	0·997	29·8	75·4	77·6	54	19
10	5·323	3·46	4·26	0·812	41·2	100·3	104·7	58	14
	4·975	3·48	4·11	0·847	37·5	93·8	95·4	60	16
12	5·513	3·33	3·81	0·874	37·6	97·7	103·4	49	12
	5·182	3·16	3·55	0·889	33·0	83·8	89·3	52	14
13	4·841	3·37	3·69	0·914	31·3	85·8	91·3	46	18
	4·771	3·00	3·63	0·827	29·6	81·1	86·6	45	17
14	2·648	4·30	5·42	0·793	26·3	68·3	75·0	50	14
	2·611	4·37	5·69	0·768	27·0	70·1	77·0		
15	4·890	3·94	4·01	0·982	37·6	87·4	98·5	58	16
	4·620	4·18	3·95	0·957	35·2	81·7	87·3	59	14

The average value for the white members of the expedition was 94·0 p.c., not significantly different from the value found by Hindmarsh (1927) for students in Sydney. In Central Australia the average was 92·0 p.c.; here these subjects had lost an average of 3·5 kg. weight.

Basal Metabolism and Relative Weight.

A number of investigators have shown the effect of rather extreme variations of the plane of nutrition of an individual on the level of his basal metabolism (Benedict, Miles, Roth, and Smith, 1919; Zuntz and Loewy, 1918; Helmreich, 1924). The demonstration of such an effect among different individuals at planes of nutrition within physiological limits emerges only from statistical analysis.

Comparable data for the relative weights and basal metabolism of 47 adult male Australian aborigines are available in the literature cited. These results give a correlation coefficient of $0{\cdot}5 \pm 0{\cdot}073$ between these values. Although few for such an analysis, the values show a definite positive correlation.

Basal Heat Loss by Evaporation.

Averages for this are shown in Table 2 and individual results in Table 4.

In the aborigines an average of 40·9 p.c. of the total heat loss was by evaporation of water from the skin and respiratory tract. The average temperature and humidity were 26·8°C. and 44·0 p.c. (10·5 mm. Hg.).

The proportion of total heat lost in this way by the whites was 29·7 p.c. in Sydney (average temperatures and humidity 24·2°C. and 53·1 p.c.), and 32·2 p.c. in Central Australia (average temperature and humidity 25·2°C. and 33·6 p.c.).

These proportions of heat lost by evaporation are higher than those found by Benedict and Carpenter (1910) (27 p.c.), and by Soderstrom and Du Bois (1914) (24 p.c.) by direct calorimetry at temperatures and humidities in the vicinity of 20°C. and 50 p.c.

The average temperatures of the present experiments were not far from that (26°C.) at which Benedict and Benedict (1927) began to observe visible sweat on their subjects. The temperatures on many occasions were above 26°C. Although the surfaces of the subjects were dry, the secretion of some sweat during the measurements is probable. This may account for the variation of some of the results.

Considerable differences were found among the white subjects in their response to change of environment. In Sydney all four evaporated water at rates within 5 p.c. of the average. In Central Australia the evaporation from the two older subjects was slightly, but not significantly, less than in Sydney. The two younger subjects increased their evaporation by about 30 p.c., and reached figures similar to those of the aborigines.

TABLE 4.

Basal Losses from Skin and Lungs.

Subject.	Date.	Lung vent. l./10 min.	R.Q.	Total insensible loss Cals. m²/hr.	Total insensible loss m²/hr.	Water Losses. Total.	Lung.	Skin.	Cals. evapd. m²/hr.	Per. cent. total.	Skin loss total evap.	Temperature C. D.B.	Temperature C. W.B.	Water vapour mm. Hg.
White Subjects.														
(1) *In Sydney, N.S.W.*														
16	9/11	39·76	0·752	37·3	20·85	20·43	4·41	16·02	11·93	32·05	78·4	22·0	15·8	10·35
	11/11	33·64	0·731	34·15	20·6	20·55	3·36	17·19	12·00	35·1	83·6	23·2	19·0	14·3
	23/11	37·65	0·788	36·2	13·75	12·90	4·10	9·76	8·09	22·35	70·5	23·0	17·2	11·75
	15/11	38·99	0·724	38·2	16·85	16·61	3·66	12·95	9·70	25·39	77·9	22·0	18·8	10·35
17	14/11	32·63	0·781	36·45	21·5	21·80	3·47	18·33	12·73	32·15	62·7	21·5	18·8	14·9
	18/11	32·09	0·835	32·25	15·1	13·30	3·57	9·73	7·77	24·1	73·2	23·3	18·2	13·1
18	9/9	45·35	0·776	33·6	29·1	28·43	4·48	23·95	16·60	49·4	84·3	17·0	9·4	4·2
	19/9	37·83	0·817	27·6	20·3	19·18	4·38	14·80	11·20	40·6	77·2	18·2	15·2	11·0
19	17/11	57·12	0·742	38·85	26·3	26·10	5·32	20·78	10·92	27·4	74·6	22·5	18·0	13·7
	22/11	54·72	0·749	39·85	18·7	18·20	4·75	13·95	12·96	32·29	77·6	23·7	16·7	10·65
	24/11	53·27	0·760	40·15	22·7	22·20	4·98	17·22	15·24	35·9	79·6	26·0	21·5	14·75
Mean		42·10	0·768	35·85	20·5	19·95	4·13	15·82	11·65	32·47	79·35	24·2	17·1	11·9
(2) *In Hermannsburg, C.A.*														
16	28/1	35·55	0·757	36·8	18·2	16·15	3·57	12·58	9·43	25·62	77·9	25·6	15·6	8·55
	1/2	38·19	0·815	37·25	15·3	14·46	4·53	9·93	8·44	22·66	68·6	25·9	16·3	9·35
17	30/1	27·49	0·797	30·7	19·7	16·85	5·79	11·06	9·84	35·83	59·7	25·3	13·9	5·5
	1/2	31·42	0·787	35·55	27·6	26·63	6·50	20·13	15·55	43·75	75·6	23·1	15·6	9·75
18	2/2	39·96	0·938	29·5	18·6	16·15	4·60	11·55	9·43	31·95	71·5	23·2	14·5	8·3
19	31/1	50·45	0·757	37·65	13·4	12·28	5·05	7·23	7·17	19·03	58·9	24·2	14·0	7·2
	8/2	49·26	0·728	36·8	17·5	17·45	5·53	11·97	10·22	27·80	68·6	28·8	15·0	6·25
Mean		38·90	0·797	34·9	18·62	17·14	5·08	12·06	10·02	28·70	70·4	25·2	16·4	8·0
Aboriginal Subjects.														
3	15/1	58·04	0·755	32·4	16·75	16·41	5·88	10·53	9·59	29·6	63·7	26·0	17·0	10·3
4	15/1	27·74	0·800	23·2	18·85	15·25	2·81	12·44	8·91	38·4	81·3	26·0	17·0	10·3
5	18/1	28·49	0·802	20·3	22·2	21·61	2·83	18·78	12·62	62·1	87·0	29·0	19·4	12·35
	27/1	46·53	0·912	29·95	20·65	18·10	4·95	13·15	10·57	35·2	72·6	26·3	17·9	11·2
	4/2	49·36	0·938	40·6	18·8	15·52	5·37	10·15	9·06	22·3	65·5	26·5	19·3	12·45
6	18/1	41·25	0·856	35·65	17·45	15·45	4·42	11·03	9·02	25·3	71·6	29·0	19·4	12·35
	24/1	33·94	0·907	25·8	17·35	15·60	3·62	11·98	9·11	35·3	76·8	26·7	20·1	13·55
	26/1	41·46	0·914	30·2	24·2	21·35	4·69	16·66	12·47	41·3	78·0	30·6	20·0	12·55
7	19/1	74·40	0·817	41·5	40·1	39·80	7·78	32·08	23·24	56·0	80·5	28·5	15·5	10·7
8	19/1	52·21	1·009	34·75	29·1	25·27	6·04	19·23	14·76	42·5	76·0	26·9	15·5	10·7
	23/1	52·24	0·883	28·6	25·8	23·90	6·42	17·48	13·96	48·8	73·1	27·0	15·5	7·75
9	20/1	44·33	0·973	30·65	21·2	18·20	5·95	12·25	10·62	34·6	67·3	27·8	15·8	7·25
	25/1	52·57	1·006	34·25	27·0	24·02	6·10	17·92	14·03	41·0	74·6	26·8	19·8	14·05
10	20/1	43·15	0·860	25·3	32·6	30·40	5·82	24·58	17·75	70·2	80·8	27·8	15·8	7·25
	25/1	54·17	0·942	37·0	34·3	30·52	5·67	24·85	17·82	48·2	81·3	26·8	19·8	14·05
	3/2	65·48	0·909	51·6	34·0	29·35	8·86	20·49	17·14	33·3	69·8	23·3	12·0	5·2
11	21/1	30·66	0·972	26·9	20·9	18·30	4·19	14·11	10·69	39·7	77·1	25·0	16·1	9·5
12	21/1	27·48	0·839	17·6	21·8	20·99	3·35	17·64	12·26	69·7	84·0	25·0	16·1	9·5
	26/1	53·47	0·882	37·0	18·8	16·38	6·93	9·45	9·56	25·8	57·8	30·6	20·0	12·55
	3/2	45·00	0·802	35·5	25·7	24·49	6·33	18·16	14·30	40·3	74·1	23·3	12·0	5·2
	9/2	61·00	1·010	40·7	28·1	23·62	7·16	16·46	13·79	33·9	69·6	25·0	18·0	11·9
13	23/1	47·71	0·827	29·6	18·6	17·56	6·14	11·42	10·26	39·6	64·9	27·0	15·5	7·75
14	24/1	26·11	0·768	27·0	17·9	18·10	2·82	15·28	10·57	39·2	84·6	26·1	20·1	13·75
15	27/1	40·87	0·973	31·7	48·7	45·70	4·90	40·80	26·69	84·2	89·3	28·3	17·9	11·1
	4/2	53·81	0·958	43·7	24·7	20·70	5·79	14·91	12·09	27·7	72·1	26·5	19·3	13·8
Mean		46·04	0·891	32·46	24·46	22·62	5·40	17·22	13·22	40·7	76·1	26·8	17·6	10·5

Relative Water Loss from Skin and Respiratory Tract.

In the aborigines the average loss from the skin was 76·1 p.c. of the total extrarenal water loss. In the whites the proportion was 79·4 p.c. in Sydney and 70·4 p.c. in Central Australia. Both these figures are higher than those found by Benedict and Benedict (1927), who showed that about half of the insensible water loss came from the skin. Most other investigators (von Czike, 1931) have found that about two-thirds of the total loss came from the skin.

No clear relation appears between external temperatures and individual losses by evaporation. When the results are considered in three temperature groups, 23–25°, 25–27°, and 27–29°C., the average heat losses by evaporation are 37 p.c., 38 p.c., and 47 p.c. of the total. Up to 27°C. the loss by evaporation seems to be on the whole unaffected by temperature, but above this there is a sharp rise, as was found by Benedict and Benedict (1927).

Curiously enough, however, the proportion of the evaporative loss from the skin remains practically the same in the three temperature groups: 73 p.c., 74 p.c., and 72 p.c. These results do not indicate that the increase of evaporative loss above 26°C. is due entirely to a sudden increase in the activity of the sweat glands; the loss by respiration shares in it equally. Any rise of body temperature which might occur in the higher temperature range could hardly be sufficient to explain the increased loss of water from the lungs in the manner suggested by the work of Jacquot and Mayer (1925) and of Christie and Loomis (1932).

No relation is obvious between the humidity of the air and the extrarenal loss of water. Osborne (1914) found that the dry bulb temperature was the determining factor in this loss at the temperatures observed.

Insensible Loss During Twenty-four Hours.

In measurements over this period all food was taken from and excreta put into sealed cans, which were weighed with the subject. All liquid drunk was measured. The volume of urine passed was usually recorded also. Table 5 gives the results obtained on four white and five aboriginal subjects. The figures include those from a white born in Central Australia and not otherwise examined (No. 20).

The insensible loss for the whites in Sydney averaged 27·6 gm./m.^{2}/hr. In Central Australia the average figure was 66·7 gm./m.^{2}/hr. This large increase is interesting in view of the small difference between the average basal losses in the two places, where the activities of the subjects were similar.

The average loss for the aborigines was 87·3 gm./m.^{2}/hr., or 29 p.c. higher than that for the whites.

Insensible Perspiration During Exercise.

During twenty-four-hour experiments activity could not be so controlled as to make results from different individuals strictly comparable. For a better

comparison between the insensible perspiration of one white and several black subjects they were weighed before and after walking together over the same route for about two hours. A similar comparison was made when the white and the black subjects were engaged for about the same period on sedentary occupations in the shade. The results of these experiments are shown in Table 6.

TABLE 5.

Insensible Loss during Twenty-four Hours; (a) Whites in Sydney; (b) Whites in Central Australia; (c) Aboriginals in Central Australia.

	Subject.	Period. hours.	Loss. gm.	Loss gm. in 24 hrs.	Loss gm. per m²/hr.	Water intake. ml.	Urine. ml.
16	White, Sydney	23·34	1,557	1,600	38·6	1,675	422
		24·62	1,410	1,375	32·1	1,380	510
17	„ „	23·16	1,021	1,058	26·6	1.090	710
		24·47	1,119	1,098	28·1	772	565
		23·78	930	935	24·0	849	1,030
19	„ „	22·80	1,188	1,251	25·3	1,774	1,632
		25·98	1,459	1,348	26·9	1.600	
		24·32	987	972	19·4	3,184	3,040
16	White, C.A.	22·42	1,765	1,686	48·9	1.790	380
		22·98	3,314	3,462	85·5	1,500	960
17	„ „	23·51	2,188	2,231	58·1	1,856	
		21·18	2,516	2,850	74·4	2,036	
18	„ „	25·30	2,957	2,806	62·8	2,000	
		24·05	4,157	4,148	92·5	2,400	
19	„ „	23·93	1,942	1,948	40·2	1,050	630
		23·67	3,550	3,600	74·7	2,340	570
20	„ „	23·47	2,922	2,990	67·7	2,300	
5	Aboriginal, C.A.	23·47	2,935	3,005	95·2	1,645	
10	„ „	23·78	3,465	3,498	95·2	2.090	
12	„ „	23·52	2,995	3,054	69·3	2,745	
21	„ „	24·23	2,916	2,890	73·0	2,100	
Means:	Whites, Sydney				27·64		
	Whites, Hermannsburg				66·71		
	Blacks, Hermannsburg				83·32		

TABLE 6.

Insensible Loss from White and Aboriginal Subjects under Comparable States of Activity, (a) Walking Exposed to Sun; (b) Resting in Shade.

	Subject.	Period. hr.	Loss. gm.	Loss. m²/hr. gm.	Temperature. D.B.	W.B.
18	(White), walking	2·96	1,408	255·5	34·4	18·1
22	(Aboriginal), walking	2·45	1,259	342·5	34·4	18·1
18	(White), resting	2·30	675	134·5	35·0	23·1
10	(Aboriginal), resting	2·02	525	163·5	35·0	23·1
9	(Aboriginal), resting	1·85	421	142·2	35·0	23·1

The loss per unit of body surface and time is 34 p.c. greater for the black than the white during the activity in the sun. The average difference for the less intense activity in the shade was only 13·5 p.c. greater for the blacks, but the activities of the subjects are not as comparable.

Effect of Solar Radiation on Insensible Perspiration.

This was measured by weighing the subjects before and after exposure to the sun for measured periods. The effect on nude and on clothed subjects was observed. The period of exposure was limited to 30 minutes, owing to the danger of burning the nude white subjects. For half the period the subjects lay with the ventral surface up, for the other half with the dorsal surface up. The upper surfaces of the body remained dry; the surfaces in contact with the ground were moist with sweat, but they dried very rapidly and were dry when the weighings were made. These measurements are given in Table 7.

TABLE 7.

Effect of Solar Radiation on Insensible Loss.

Subject.	Period.	Loss.	Loss. m^2/hr.	Increase during 30′ exposure. m^2/hr.	Mouth temp.	Wet bulb.	Dry bulb.
	hr.	gm.	gm.	gm.	C.	C.	C.
19	0·915	107	57·9		36·65	23·2	45·2
White, nude	0·965	105	53·9				
	1·015	658	320·5	529·2	36·9		
	0·98	145	73·3				
	0·98	135	68·2		36·65		
9	1·06	116	66·7		36·8	23·2	45·2
Black, nude	1·01	89	53·7				
	1·03	595	352·0	585·6	37·15		
	1·085	123	69·1		36·8		
18	0·97	196	108·5		36·3	29·0	52·5
White, nude	1·02	550	289·6	362·2	36·8		
6	1·08	135	78·6		36·8		
Black, nude	0·92	523	258·6	560·0	36·9		
19	1·02	166	80·6		36·8	29·0	52·5
White, clothed	1·13	547	239·6	318·0	36·95		
12	0·98	120	74·2		36·55	29·0	52·5
Black, clothed	1·23	420	207·1	265·8	37·15		

The increase of insensible perspiration on insolation has been calculated on the assumption that the whole of the increase occurred during the period of exposure. This is an approximation, but experiments in which after-periods were measured show that the insensible perspiration fell very rapidly towards its first value on taking shelter. Also this period of falling loss was compensated by the period of rising loss at the beginning of the exposure.

The average increase for the nude whites was 445·7 gm./$m.^2$/hr., and for the nude blacks 572·8 gm./$m.^2$/hr., or 29 p.c. higher. It is rather suggestive that the difference between the insensible perspiration of the whites and blacks under these conditions, walking and resting in the sunlight, and under basal conditions, was about 30 p.c., and that Martin (1930) found that black skin absorbed heat from the sun's ray about 30 p.c. faster than white skin.

For the clothed subjects the increase of insensible perspiration for the white on exposure to the sun was 318·0, and for the black 265·8 gm./$m.^2$/hr. The

increase is here much less; the difference between the black and the white is also much less, and is in the opposite direction.

The extra heat dissipated by the subject when exposed to the sun was evidently slightly less than that absorbed, as the mouth temperatures rose slightly. Although mouth temperatures are not reliable indices of general body temperature, it may be assumed that the rises of mouth temperature are associated with a general rise of body temperature.

SUMMARY.

The average basal metabolism of 10 male Central Australian aborigines examined immediately after leaving their natural surroundings, was 82 p.c. of the Aub-Du Bois standards. Four white subjects examined at the same time gave a value of 92 p.c. of this standard; this value is not significantly different from that for the same subjects in Sydney.

The weight of the aboriginal subjects averaged 88·4 p.c. of the standards of weight for height and age used for comparison. The average weight of the white subjects was 100·9 p.c. of these standards while in Central Australia and 106·2 p.c. while in Sydney.

Under basal conditions evaporation of water from the skin and respiratory surfaces accounted for 40·9 p.c. of the total heat lost by the aborigines, and for 29·7 p.c. of the total in the whites; in Sydney their figures were not significantly different.

Evaporation from the skin was 76·1 p.c. of the total for the aborigines and 70·4 p.c. for the whites in Central Australia. In Sydney the figure for the latter subjects was 79·4 p.c. of the total.

In contrast with their basal loss, the white subjects during 24 hours evaporated more than twice as fast in Central Australia as they did in Sydney under similar conditions of activity (66·7 gm./m.^{2}/hr. and 27·4 gm./m.^{2}/hr. The average rate of evaporation from the aboriginal subjects was 83·3 gm./m.^{2}/hr.

The average loss of heat by evaporation rose abruptly when the environmental temperature rose to 27°C. (38 p.c. of total up to 27°C., 47 p.c. of total 27°C.–29°C.). The proportion of skin loss to lung loss remained unchanged.

During exposure to the sun at rest, nude black subjects increased their loss by evaporation 28·5 p.c. more than nude white subjects. The increases were in the vicinity of twenty times the basal values (external temperatures 45·2°C.–52·5°C.). The wearing of clothing considerably diminished the extra evaporation of both subjects and abolished the advantage of the black subject.

During activity a black subject exposed to the sun increased his rate of evaporation about 30 p.c. more than a white subject.

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THE VOLUMETRIC MICRODETERMINATION OF POTASSIUM AS POTASSIUM ACID TARTRATE IN BIOLOGICAL MATERIAL

by

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The material under investigation is digested with perchloric acid, nitric acid, and hydrogen peroxide, and the potassium is isolated as potassium perchlorate. After dissolving the potassium perchlorate, the potassium is reprecipitated with an alcoholic solution of tartaric acid as potassium acid tartrate. The potassium acid tartrate is titrated against 0·01N sodium hydroxide.

REAGENTS.

1. 60–70 p.c. perchloric acid, chemically pure.
2. Concentrated nitric acid, chemically pure.
3. 30 p.c. hydrogen peroxide, chemically pure.
4. A mixture of equal parts of n-butyl alcohol and ethyl acetate.
5. A cold saturated alcoholic solution of tartaric acid. This solution is prepared by shaking 3 gm. of ground crystals of tartaric acid in 100 c.c. of absolute alcohol for about 15 minutes, followed by filtration. This solution must not be kept longer than two weeks.
6. 0·01N sodium hydroxide.
7. A composite indicator consisting of 1·25 gm. methyl red and 0·05 gm. methylene blue dissolved in 100 c.c. alcohol.

Digestion of Material and Isolation of Potassium as Potassium Perchlorate.

As described in a previous communication (Bolliger, 1932) the material is digested with perchloric acid, nitric acid, and hydrogen peroxide. Next, nearly all of the perchloric acid is evaporated, and all the perchlorates formed with the exception of potassium and ammonium perchlorate, are extracted with a mixture of butyl alcohol and ethyl acetate. The ammonium perchlorate, most of which is formed in the process of digestion, is then removed by heating (whereby partly it decomposes into nitrogen, chlorine, and water).

If possible, the whole of the analysis should be performed in a tube which can be centrifuged. An actual centrifuge tube, about 13 cm. in length and 1·2 cm. in width, covered with a small funnel during the process of digestion, was found to be satisfactory for small samples, such as 0·4 gm. of wet tissue or 1 to 2 c.c. of fluid.

When working with material difficult to digest, in the course of a potassium determination, the vessels used for the digestion of the organic material should preferably be of silica.

In the previous communication (Bolliger, 1932) the rapidity of the ashing with perchloric acid was emphasized, and it was shown how the digestion could be completed in less than half an hour by heating directly over the flame. In the present case, particularly if the material contains much nitrogen or is difficult to digest, as with fatty tissue, or if a series of determinations is done simultaneously, a slower technique of digestion is recommended. It is advantageous in such cases to let the material undergo a preliminary digestion with nitric acid at water bath temperature, then to add the perchloric acid and keep this nearly or actually boiling with little evaporation for an hour or more. A glycerine bath (glycerine and sodium nitrate) or a tri-cresyl phosphate bath heated over a gas flame or hot plate was found very useful for keeping perchloric acid at slow boiling temperature. However, vapours from the oil bath have to be prevented from getting into the centrifuge tube. This technique of comparatively slow ashing requires less personal attention and, since the destruction of organic matter is more complete, violent explosions are avoided during the destruction of ammonium perchlorate. For this modified technique we give as an example the ashing of milk, a substance which is fairly difficult to digest.

In a centrifuge tube (about 1·2 x 13 cm.) 1 c.c. whole milk and 2 c.c. of nitric acid were heated in a boiling water bath for about an hour. Then after the addition of 2 c.c. of 70 p.c. perchloric acid, the tube, covered with a small funnel, was transferred to the oil bath, which was slowly heated with a small flame. At first the remaining nitric acid was evaporated, and then the perchloric acid began to boil and to evaporate to a small extent. At the end of 3 hours about 0·5 c.c. of perchloric acid still remained. Then about 0·5 c.c. of 30 p.c. hydrogen peroxide was carefully added, drop by drop, in order to destroy the bulk of the organic matter still present. After further heating, till practically only a trace of perchloric acid remained, the centrifuge tube was taken out of the oil bath and cooled. 3 c.c. of a boiling mixture of equal parts of n-butyl and ethyl acetate were then added, and the centrifuge tube was cooled again in tap water, and finally in the ice chest, before centrifuging out the undissolved salt mixture, consisting of potassium and ammonium perchlorate. The supernatant fluid containing all the other perchlorates was drained off and the mouth of the inverted centrifuge tube was rinsed with alcohol. For the determination of any of the perchlorates in solution, the extraction with butyl alcohol and ethyl acetate has to be repeated.

In order to expel the last traces of the butyl alcohol mixture the centrifuge tube is again put into the hot oil bath for a few minutes. It is then carefully heated over a small flame. Sometimes a fairly sudden decomposition and the formation of chlorine gas indicates the destruction of most of the ammonium perchlorate. To assure its complete removal the entire tube has now to be brushed thoroughly with a moving flame. There is little fear of loss, because any potassium perchlorate which is decomposed will break down to potassium chloride and oxygen. In case the ash obtained should not be perfectly white an additional

treatment with 0·5 c.c. of boiling perchloric acid, and the subsequent addition, drop by drop, of hydrogen peroxide will rectify this matter.

Sera and small pieces of tissue of less than 0·5 gm. have been treated similarly, but such substances as blood filtrates, gastric juice, and urine, which are easily digested, may equally well be ashed as described in the previous communication.

If no sufficiently large centrifuge tube is available, a silica beaker of 100–200 c.c. capacity, covered by an inverted funnel of similar diameter, was found to be suitable for larger samples of tissue amounting up to several grams. After the preliminary digestion with nitric acid in the water bath, perchloric acid is added in an amount of about four times as many cubic centimetres as there are grams of wet tissue present. The beaker is put in the glycerine bath, which is kept at such a temperature that the perchloric acid barely evaporates through the funnel stem. After contact with the hot perchloric acid has been maintained for several hours, the evaporation of the perchloric acid is speeded up by removing the funnel. When only about 0·5 c.c. of perchloric acid remains, about 1 c.c. of hydrogen peroxide is added drop by drop. After evaporating nearly all of the perchloric acid, the remaining ash should consist of white crystals of moderate size, which will allow the decantation of the butyl alcohol-ethyl acetate mixture, which is added as described before. The addition of approximately 2 c.c. of the butyl alcohol mixture for every mg. of soluble perchlorates expected to be present is sufficient.

The Reprecipitation of the Potassium as Potassium Acid Tartrate and its Titration.

The potassium perchlorate is completely dissolved in a sufficient amount of warm water. Then to each volume of the perchlorate solution at least 10 volumes of cold saturated alcoholic tartaric acid are added. With a thin glass rod the solutions are mixed and immediate crystallization will occur. The mixture, after standing in the ice box for at least 4 hours is centrifuged, and the supernatant fluid is drained off. Then the crystals of acid potassium tartrate are washed twice with about 3 volumes of absolute alcohol, and 1–2 c.c. of water are added. Samples which have not been digested in a centrifuge tube may be transferred into such before precipitating with tartaric acid. However, it is also possible to form the tartrate in the original container and to decant through a filter in order not to lose any floating particles. Repeated washings with absolute alcohol are necessary in order to remove all traces of tartaric acid. Any particles of bitartrate on the filter are dissolved with hot water, the solution being added to the bulk of the precipitate. A few drops of the composite indicator, consisting of 1·25 p.c. of methyl red and 0·05 p.c. of methylene blue dissolved in alcohol, are added. During titration against 0·01N sodium hydroxide the contents of the centrifuge tube or beaker are stirred with a glass rod. The appearance of a distinct green colour is taken as the end point.

DISCUSSION.

As pointed out previously, we consider digestion with perchloric acid advantageous. However, according to the material to be analysed, as well as the laboratory conveniences available, the technique has to be modified as suggested in the experimental part. The separation of the potassium perchlorate from the other perchlorates is similar to the modification of the method of Schlösing-Wense, as given by Treadwell (1930). As in the case of potassium chloride, tartaric acid precipitates the potassium of a potassium perchlorate solution as potassium bitartrate. With an alcoholic tartaric acid solution the precipitation is sufficiently complete and specific for the usual purposes of micro-analysis even in the presence of a small amount of the sodium salt. Experimentally it was found that one part of sodium perchlorate in five parts of potassium perchlorate did not interfere with the determination. Therefore, for the potassium determination itself, the sodium perchlorate has not to be extracted quantitatively. Also, the formation of some potassium chloride, while destroying the ammonium perchlorate, does not interfere. Tartaric acid in the cold forms small amounts of its mono ethyl ester with ethyl alcohol. This does not influence the precipitation of potassium if the tartaric acid solution is prepared as described. As solutions become older they sometimes throw down a partly colloidal precipitate. Tartaric acid dissolved in acetone gave values which were somewhat too low.

In the double precipitation method described, the presence of moderate amounts of sulphates or phosphates is of no importance. The greater part of these acids are extracted with the mixture of butyl alcohol and ethyl acetate, and small amounts of sodium sulphate that may be present are apparently dealt with satisfactorily by the second precipitation with tartaric acid.

With the method described, results obtained agree closely with those obtained by macro-analysis with the chloroplatinate method. In the case of cow's milk, 140, 142, and 144 mg. p.c. were found to be present, and 100 mg. of potassium added to 100 c.c. of milk were recovered with an error of $\pm$ 2 p.c.

SUMMARY.

A method is described for the volumetric determination of potassium in biological material as potassium bitartrate, subsequent to its isolation as potassium perchlorate.

ACKNOWLEDGMENTS.

I desire to express my sincere thanks to the Board of Directors of the Sydney Hospital for extending to me the facilities necessary for the completion of this investigation, and to Dr. Keith Inglis and Dr. H. I. Wardlow for their sympathetic assistance in arranging for these facilities to be available. My thanks are also due to Miss Dorothy Dark, who assisted me in this investigation.

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THE RESPIRATORY EXCHANGE OF THE AUSTRALIAN ABORIGINE

by

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(Submitted for publication 10th May, 1934.)

In a previous paper (1933) an account was given of the results of oxygen uptake and surface area measurements on natives living in a state of nature in the area west of Cockatoo Creek, in tropical Central Australia. We now report results obtained during the expedition from the University of Adelaide to Mount Liebig, some 250 miles west of Alice Springs, Central Australia.

From experienced gained at Cockatoo Creek it seemed not unreasonable to attempt to estimate the total gaseous exchange on native subjects over a period of at least six hours, in the hope that some insight might be gained into the nature of the foodstuffs oxidized. It was hoped that we might experience the same diurnal extremes of temperature as those met with at Cockatoo Creek, but unfortunately the night temperature never sank so low. It was found possible to persuade natives to subject themselves to a basal metabolism determination in the early morning, about 6 a.m., and, in addition, to remain recumbent by their camp-fires until 1 p.m., so that at intervals expired air could be collected by the Douglas bag method and analysed. The practical difficulties in the way of conducting such work in the field are considerable, quite apart from the difficulty of conveying instructions by signs or through interpreters of mediocre capacity. The roughness of the transport destroyed our duplicate Haldane apparatus, the heat and dryness worked disaster with rubber connections, and our katharometer was completely thrown out of action.

All gas analyses were performed in triplicate, and the results agreed satisfactorily among themselves. The Benedict field apparatus was used as before for the early morning measurement of basal oxygen requirement, and at 8 a.m. a portion of almost raw meat was given to one of the two subjects, the other remaining in fasting condition.

Skin temperatures were measured by means of special flat-bulbed thermometers, kindly lent by Sir Charles Martin, and, whenever possible, measurements were made of air and sun temperatures, and of the rate of cooling by means

of the katathermometer. In this we were greatly assisted by Mr. E. Holden, a member of the anthropological party. Had we been certain that the natives would co-operate to the extent that they did, our field preparations would have been more extensive, but enough has been done to indicate the value of this type of investigation. Essential factors are: (1) The natives were entirely uncontaminated or influenced by contact with the white man, and lived naked, a nomadic life

Benedict Field Metabolism Apparatus as used in Central Australia.
Note the thermometers in the axillae and fires at points marked X.

in the arid and desert country west and north of Mount Liebig; (2) they sleep on the ground near small camp-fires, with a low wind-screen of about 2 feet 6 inches of brushwood behind them; (3) the N'galia tribe subsisted mostly upon roots, etc., living as they do in the sandhill country bordering Mount Liebig; (4) the Pintobe tribe came from a small range of hills near the West Australian border, where water, and therefore game, was obtainable. They looked, despite their long trek, physically superior to the N'galias; (5) the natives experimented upon were under surveillance throughout the period from 6.30 a.m. to 1 p.m.; (6) No satisfactory dietetic history was available, except the fact that the subjects were, so far as our evidence could go, in the post absorptive state (it is not possible to be certain on this point); (7) owing to drought conditions the N'galias at least were definitely on the border of under-nutrition.

Cockatoo Creek, August, 1931.

Corrected Values for Oxygen Consumption and Derived Energy Production at an Oxygen Calorific Equivalent of 4·825 Calories per litre.
See this Journal, Vol. 11, 1933, p. 181, Table 1.

No. of Expt.	Minimum O_2 per min.	Calories per hour.	Calories per hour Predicted.	Calories per sq. metre per hour.	Deviation.
64	215	62	60	33·5	+3%
64A	246	70·8	65·4	40·2	+6%
65	203	58·8	54·3	40·5	+8%
65A	210	60·8	63·4	36·6	—4%
66	165	47·8	64·5	18·5	—26%
66A	210	60·9	63·4	41·0	—8%
67	205	59·3	59·5	31·5	±0%
67A	211	61·1	53·3	42·7	+14%
68	218	63·1	55·0	40·8	+14%
68A	222	64·3	62·3	40·2	+3%
69A	226	65·4	59·8	40·2	+9%
70A	213	54·3	52·6	33·1	—2%
71	192	57·9	62·4	34·6	—8%
71A	211	61·1	53·6	37·1	+14%
72	168	48·9	51·4	34·0	—5%
72A	232	67·2	59·0	41·2	+14%
73	180	52·1	66·0	26·4	—20%
73A	240	69·4	60·8	43·9	+12%
74	173	50·1	51·5	34·6	—3%
74A	178	51·5	57·3	35·2	—10%
75	217	62·8	60·0	36·5	+4%
76	180	52·1	56·0	34·8	—7%
77	185	53·1	50·4	39·9	+5%
78	192	55·6	65·0	35·5	—14%
78A	180	52·1	51·1	38·5	+2%
79	171	49·5	62·0	28·4	—20%
79A	202	58·5	56·0	35·5	+4%
80	193	55·9	55·6	34·9	±0%
81	221	63·9	57·0	36·1	+12%
82	201	58·2	56·7	39·6	+3%
83	168	48·6	54·1	34·5	—10%
84	174	50·4	54·9	31·6	—9%

From the Cockatoo Creek results it seemed that excessive heat loss resulted from the exposure to cold during the nights. From this, and from the low figures for oxygen consumption obtained in mid-summer at Koonibba (1929), it was thought desirable to study the metabolism continuously, from cold early morning to hot midday, when it was anticipated that a falling metabolism would disclose itself. A *slight* fall was found, but the outstanding point of interest is the general trend of the values for the respiratory quotient. Moreover, since conducting these experiments, a lost Cockatoo Creek field notebook, from which entries had been transcribed to a Carnegie Nutrition Laboratory book, has been found, which makes it clear that a systematic error in transcription of logarithms without mantissae, was made in the field, the logarithm itself giving no indication of a wide departure from the true value. The recalculated results,

which are quoted in this paper, indicate practically no effect of heat loss, as previously reported, and thus the present basal metabolism results, though obtained in much warmer night conditions, are what might have been anticipated. The respiratory quotients, however, show, not only some very low values, but also a rather rapid trend from high to low values during the morning fast. The charted results show the R.Q. values for the fed and the fasting native side by side. Despite the experimental difficulties and consequent tendency to error throughout, and despite the insufficiency of the dietetic history, it will be seen that the basal metabolism of the fasting subject either remains unaltered, or tends to fall, and that the fasting R.Q. does likewise.

Collecting Douglas Bag sample. Note brushwood shelter with spears lying thereon, and fires at points marked with arrows.

This latter may be a result of the fact that these subjects live near the lower margin of normal nutrition. The fall in basal oxygen requirement does support the conclusion drawn from the Koonibba observations, namely, that the low values there obtained were, at all events in part, due to summer temperatures, under-nutrition also contributing thereto.

The specific dynamic effect of the meat protein intake is greater than that reported by Benedict and Carpenter (1918), with whose experiments these most closely agree in details. The immobility of the subject can be vouched for, and

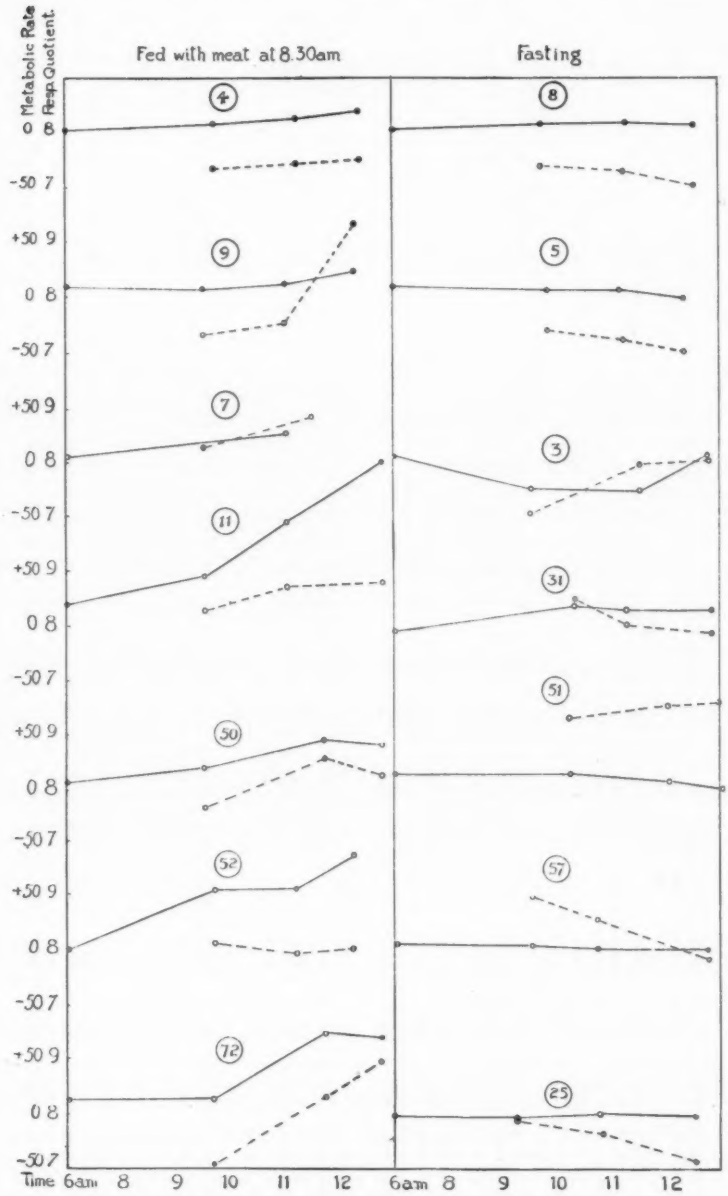
Metabolic Rate
Resp. Quotient.
Fed with meat at 8.30am
Fasting
4
8
9
5
7
3
11
31
50
51
52
57
72
25
0 8
-50 7
+50 9
Time 6am 8 9 10 11 12 6am 8 9 10 11 12

Fig. 1.

TABLE 1.

No.	Time.	c.c. O_2 per min.	Cal. per hr.	Cal. per 24 hrs.	Cal. per 24 hrs. Calculated.	Deviation.	Tidal Air.	R.Q.	Pulse.	Respn.	Temp. of Body. Axill.	Face.	Chest.	Abdomen.	Temp. Black Velvet.	Air Temperature. Wet.	Dry.	Sun.	Kata Cooling Power. Wet.	Dry.	Relative Humidity.	Max. °C.	Min. °C.
10/8/32																						21·6	14·6
4 B	7.0 a.m.	224·0	63·0	1,514	1,483	+2	Fed		60–60	15–16	36·3	29·0	27·0	27·0			13·5						
4 DB 1	9.40 a.m.	279·2	79·3	1,613		+9	353	·734	96–96	14	37·1	35·5	34·2	34·5		10·8	14·6				63		
4 DB 2	11.5 a.m.	293·4	83·2	1,691		+14	218	·740	96	24	37·1	36·0	34·0	36·0									
4 DB 3	12.25 p.m.	314·8	89·3	1,815		+22	316	·746	100	16–18	36·7	36·0	37·0	38·5		10·0	18·3				26		
8 B	7.20 a.m.	222·5	63·1	1,515	1,480	+2	Fasting		45–51	16–16	35·8	35·5	33·2	33·0		10·8	14·6				63		
8 DB 1	9.40 a.m.	237·5	67·4	1,617		+9	282	·737	54	18	37·1	34·7	32·0	34·0									
8 DB 2	11.10 a.m.	256·8	72·6	1,742		+10	253	·727	66	18	37·6	37·0	32·0	35·0		10·0	18·3				26		
8 DB 3	12.30 p.m.	235·0	66·1	1,586		+7	228	·700	61	21	37·1	36·7	37·0	36·0									
11/8/32																							
9 B	7.0 a.m.	237·3	67·1	1,610	1,442	+10	Fed		55	16	36·2	31·5	26·0	25·0			9·0		3·3	14·2	50	15·1	9·0
9 DB 1	9.30 a.m.	229·4	64·88	1,557		+8	337	·732	69	16–12	38·0	38·0	35·2	35·2		8·3	12·7						
9 DB 2	11.0 a.m.	237·9	67·8	1,627		+12	354	·764	69	12–12	37·8	37·8	37·2	36·5									
9 DB 3	12.15 p.m.	249·4	74·2	1,776		+23	355	·932	68	14–14	37·8	34·0	34·5	39·0		9·7	15·5		38·5	16·0	41		
5 B	7.0 a.m.	374·5	67·5	1,620	1,476	+9	Fasting		69	18	36·6	32·5	27·5	29·0			9·0						
5 DB 1	9.50 a.m.	228·2	64·7	1,552		+5	263	·739	90	18	37·2	38·0	37·0	38·0									
5 DB 2	11.5 a.m.	229·2	64·6	1,350		+5	284	·722	78	18	37·2	38·0	37·2	38·0									
5 DB 3	12.15 p.m.	213·1	59·9	1,437		—2	221	·700	78	18	36·9	36·0	36·5	38·5									
12/8/32																							
3 B	7.0 a.m.	207·0	58·2	1,183	1,136	+4	Fed		45	12	35·5	31·0	28·0	2 ·			6·0		38·2	27·0	37	17·9	1·1
3 DB 1	9.30 a.m.	135·6	38·1	912		—24	189	·706	48	14	35·3	33·8	33·0	3· 0									
3 DB 2	11.25 a.m.	162·0	37·0	888		—27	251	·790	56	14	37·0	37·0	35·3	36·0									
3 DB 3	12.45 p.m.	172·8	49·9	1,200		+5	260	·802	54	14	37·5	35·0	35·5	35·0		8·3	16·6		35·0	11·5	22		
7 B	6.0 a.m.	221·4	67·9	1,629	1·568	+4	Fasting			11		34·5	30·8	29·5									
7 DB 1	10.0 a.m.	293·4	86·3	2.070		+32	480	·88	66	11	37·8	35·0	37·0	37·0									
13/8/32																						19·6	6·7
10 B	6.0 a.m.	294·2	84·5	1,719	1,238	+38	Fasting*		78	15	36·7	29·0	30·5	32·0			6·0						
10 DB 1	9.30 a.m.	214·4	61·6	1.478		+19	246	·789	70	18	36·9	32·0	33·5	32·5	23·0	6·1	10·5	18·0			46		
13 B	6.45 a.m.	255·7	75·2	1,529	1,136	+34	Fed		60	16	36·2	31·5	30·0	30·0	35·8			21·5					
13 DB 1	9.45 a.m.	257·1	75·6	1,814		+60	349	·883	60	16	36·9	38·0	38·2	38·0		6·1	10·5				46		
14/8/32																							
31 B	6.30 a.m.	228·0	66·5	1,353	1,441	—6	Fasting		54	18	36·7	30·0	29·2	30·0			8·7						
31 DB 1	10.15 a.m.	245·3	71·5	1,716		+18	271	·846	60	18	37·4	32·0	33·5	34·0	20·0			17·2					
31 DB 2	11.15 a.m.	240·0	69·1	1,659		+14	327	·799	62	18	36·9	39·3	39·0	39·2									
31 DB 3	12.45 p.m.	241·0	68·9	1,653		+14	288	·775	73	19	37·5	38·5	38·0	36·5									
11 B	7.0 a.m.	229·6	66·5	1,596	1,329	+20	Fed†		57	17	36·3	30·0	27·0	29·0			8·7						
11 DB 1	9.30 a.m.	281·6	81·7	1,960		+47	342	·828	57	15	37·0	32·0	33·5	34·0					30·5	22·0	46		
11 DB 2	11.0 a.m.	381·2	108·9	2,610		+96	467	·867	61	19	37·3	38·5	39·5	39·0	28·5			21·0					
11 DB 3	12.45 p.m.	487·4	139·6	3,336		+150	473	·879	63	19	37·3	36·2	35·8	37·5					35·0	16·0	34		

* Subject ill at ease.
† Subject apparently calm and comfortable.
Note: B = Benedict Apparatus; DB = Douglas Bag.

TABLE 1 (contd.)

* Subject ill at ease.
† Subject apparently calm and comfortable.
Note: B = Benedict Apparatus; DB = Douglas Bag.

TABLE 1 (contd.)

No.	Time.	c.c. O_2 per min.	Cal. per hr.	Cal. per 24 hrs.	Cal. per 24 hrs. Calculated.	Deviation.	Tidal Air.	R.Q.	Pulse.	Respn.	Temp. of Body: Axill.	Face.	Chest.	Abdomen.	Temp. Black Velvet.	Air Temperature: Wet.	Dry.	Sun.	Kata Cooling Power: Wet.	Dry.	Relative Humidity.	Max. °C.	Min. °C.
15/8/32																							
50 B	6.30 a.m.	220·0	63·4	1,521	1,406	+8	Fed		64	18	37·4	34·0	31·0	30·0	8·0		8·0					16·8	3·9
50 DB 1	7.30 a.m.	247·0	70·8	1,702		+21	300	·769	61	20	37·4	36·0	34·0	34·0		2·8	7·8		37·0	20·0	43		
50 DB 2	11.40 a.m.	287·0	84·0	2,015		+44	535	·854	61	12	37·5	—	37·0	38·0	29·5			24·5					
50 DB 3	12.45 p.m.	260·0	82·0	1,966		+40	426	·827	76	16	37·7	35·5	35·0	38·0	29·2			37·0			49		
15/8/32																							
51 B	7.15 a.m.	288·3	83·7	2,008	1,739	+15	Fasting		54	18	36·1	29·5	27·5	26·5			8·0						
51 DB 1	10.15 a.m.	268·8	78·0	1,872		+15	236	·929	73	27	36·6	38·0	37·0	35·0	23·5			18·0					
51 DB 2	12.0 p.m.	320·3	95·8	2.299		+7	390	·949	66	26	36·6	36·5	36·0	36·5					43·0	21·0	36		
51 DB 3	1.0 p.m.	236·4	70·89	1,701		±0	346	·961	66	22	37·8	39·5	37·5	37·5									
16/8/32																							
57 B	6.45 a.m.	260·3	76·0	1,564	1,450	+8	Fasting																
57 DB 1	9.30 a.m.	216·6	64·0	1,537		+6	357	·904	84	20	36·8					2·7	8·6		30·6	16·0	26	21·8	3·4
57 DB 2	10.47 a.m.	206·6	60·4	1,450		±0	577	·862	69	12	36·3												
57 DB 3	11.45 a.m.	205·0	59·2	1,421		—2	317	·792	76	17	37·2					5·5	15·3		32·0	11·2	7·5		
16/8/32																							
52 B	6.30 a.m.	241·5	69·8	1,419	1,429	±0	Fed		70	14	36·0						6·0						
52 DB 1	9.40 a.m.	317·4	91·62	2,198		+53	444	·814	75	14	36·0					2·8	8·6		30·6	16·0	26		
52 DB 2	11.12 a.m.	317·7	91·5	2,196		+53	403	·797	90	14	36·1					5·5	15·3		32·0	11·25	7·5		
52 DB 3	12.15 p.m.	383·8	110·6	2,640		+84	409	·804	110	18	37·5												
17/8/32																							
25 B	7.0 a.m.	218·8	63·0	1,512	1,508		Fasting		43	11	35·6	29·5	29·5	27·5			6·0					20·2	1·1
25 DB 1	9.15 a.m.	211·5	60·9	1,464		—3	352	·795	43	12	36·2	33·5	34·0	34·0		3·0	8·6		31·0	14·9	29		
25 DB 2	10.50 a.m.	224·5	64·0	1,536		+1	557	·773	48	10	36·6	34·0	34·0	37·0									
25 DB 3	12.30 p.m.	227·3	64·0	1,536		+1	287	·726	52	20	37·7	37·2	39·0	38·5	27·0	6·4	16·7	42·0	33·3	14·0			
17/8/32																							
72 B	7.0 a.m.	218·6	63·0	1,512	1,314	+15	Fed		64	20	36·6	28·0	28·5	28·0			6·0						
72 DB 1	9.45 a.m.	325·4	91·6	2,198		+67	308	·709	66	18	36·6	34·5	38·0	38·5	26·25			21·0					
72 DB 2	11.45 a.m.	352·2	102·4	2,448		+86	308	·836	90	22	37·8	37·0	39·5	40·0									
72 DB 3	12.45 p.m.	343·1	101·4	2,424		+83	417	·899	86	17	37·8												
18/8/32																							
70 B	6.30 a.m.	186·8	52·5	1.240	1,260	±0	Fed		52	10	35·1	28·2	29·9	32·4			5·0					20·2	0·6
70 DB 1	9.50 a.m.	239·1	67·2	1,608		+27	441	·703	54	10	36·1	37·2	35·0	37·0		4·7	10·5						
70 DB 2	12.0 p.m.	252·8	73·6	1,764		+40	482	·843	72	10	37·2	39·0	40·0	39·0									
18/8/32																							
66 B	6.30 a.m.	234·4	68·2	1,632	1,445	+12	Fed		54	12	35·9	34·5	32·7	33·0									
66 DB 1	9.0 a.m.	392·9	114·4	2,736		+89	421	·842	92	18	36·3	34·2	37·2	38·0									
66 DB 2	11.0 a.m.	383·4	110·5	2,640		+82	429	·830	94	18	38·5	36·0	35·0	36·0									
18/8/32																							
67 B	6.20 a.m.	154·0	43·9	1,054	1,410	—25	Fed		51	12	35·4	29·0	27·0	28·0			5·0						
67 DB 1	10.0 a.m.	240·2	68·3	1,633		+15	638·7	·747	52	10	36·1					4·7	10·5		35·3	14·5	30		
67 DB 2	11.0 a.m.	287·4	82·3	1,969		+39	485	·775	66	12	37·8	34·0	38·0	37·5	39·0			25·0	26·6	8·8	1		

Note: B = Benedict Apparatus; DB = Douglas Bag.

in this respect the subject appears to be ideal. Overbreathing can be excluded, for in a total of 100 separate Benedict estimations of 45 minutes' duration we have never found it to occur. It may be, however, that with such a race it is possible to mask muscular tone to an extent impossible with a European—we have seen them submit to ceremonial mutilation, for example, with absolutely no sign of pain perception, and Cleland timed the pulse rate of a native during circumcision, and found that it never altered. Benedict and Carpenter's results with beefsteak showed a maximum of 25 p.c. increase between the 4th and 5th hours. Their minimum response was + 8 p.c. at the second hour.

What is even more significant is the rise in the R.Q., which not infrequently approaches unity about the 5th hour. Both the total increase in oxygen consumed, and the sharp rise in R.Q., indicate a rapid response on the part of both the tissues and the glands influencing metabolism.

The whole experiment aimed at finding if any deficiency in the specific dynamic response existed, in order that light might be thrown upon a low metabolism—if such could be proved to be normal. So far as these experiments take us, after allowance is made for error due to circumstances, there is certainly no evidence of either a smaller or slower specific dynamic response. The low blood pressure recorded confirmed the conclusion drawn from the Cockatoo Creek experience, that the vascular system offered a field for investigation, and the account of such work conducted during 1933 will shortly be published. It gives grounds for assuming more effective vasomotor control than that possessed by the civilized individual, and consequently a smaller skin circulation in the cold. Such skin temperatures as are here recorded indicate a steady rise parallel with increasing sun temperature. Blood temperature also rose, in some instances two degrees, although the katathermometer readings indicated that there was high cooling power throughout the experimental period both by radiation and evaporation. Even on the morning of 12/8/32, with a relative humidity of 50 p.c., the dry cooling power was 14 milli-calories per sq. cm. per sec. The effect of air movement on heat loss due to evaporation is well shown on that same day, when at noon the relative humidity was still 41 p.c., but the cooling power for the wet kata had risen from 3·3 to 38·5. The general appearance of the skin is misleading, so far as evidence of sweating is concerned, but actually sweating is profuse, and runs off in streams where the skin presses either against adjacent skin or the earth on which the native lies. Thus the axilla runs with gross sweat. The shoulder blades, the natal fold, the nates where they touch the ground, and the underside of the thighs are always wet when the native rises from lying in the sun; and if he is covered by a piece of sack he becomes drenched in sweat. As soon as he moves about all signs of moisture disappear, and the katathermometer readings provide the explanation. It is necessary to emphasize this feature, since it has been thought that the Australian aboriginal might have a water metabolism specially adjusted to his arid environment. Our experience of a midday ascent

of Mount Liebig—2,000 above the camp—showed that water discipline must play the main rôle, for although the natives accompanying us showed no especial interest in the water-bags, yet, when offered the water, they consumed twice as much as we did.

TABLE 2.

Field No.	Tribe.	W.	Stature (H.).	Sitting Height (S.H.).	S.H.	Pelidisi $\frac{\sqrt{W} \times 10}{S.H.} = 100$	Age.	Surface Area. Estimated from Height/Weight. Nomogram (Du Bois).	Calculated from Linear Formula (Du Bois).	Blood Pressure (Recumbent).
1	N'galia	45·6	164·5	83·3	+1·1	92·4	17	1·47	1·46	105/60
2	,,	48·6	167·5	82·0	—1·0	96·0	—	1·55	1·54	100/60
3	,,	46·5	167·4	83·7	0·0	92·5	60	1·45	1·47	102/85
4	,,	53·2	169·0	84·2	—0·3	96·3	25	1·59	1·51	112/76
5	,,	55·0	171·0	85·2	—0·3	94·2	30	—	—	105/65
7	,,	64·5	177·7	87·0	—1·8	99·3	40	1·80	1·83	100/68
8	,,	53·5	169·2	82·2	—2·4	99·0	24	1·61	1·54	118/68
9	,,	54·5	163·2	82·7	+1·3	98·1	28	1·56	1·57	106/68
10	Pintobe	48·2	176·1	87·1	—0·9	90·1	55	1·59	1·56	112/74
11	,,	55·2	168·6	83·6	—0·7	98·1	50	1·61	1·58	116/78
13	,,	46·5	172·0	85·5	—0·5	90·8	40	1·53	1·66	113/74
25	,,	53·3	170·4	83·6	—1·6	97·0	18	1·61	1·67	108/59
31	N'galia	56·5	175·2	85·2	—2·4	95·7	40	1·66	1·61	114/76
34	,,	—	—	—	—	—	—	1·57	—	—
50	Pintobe	55·2	170·1	82·4	—2·6	97·5	40	1·63	1·55	102/68
51	Luritja	62	173·2	81·3	—5·3	104·9	60	1·75	1·71	119/82
52	N'galia	51	170·1	84·0	—1·0	95·2	28	1·57	1·56	123/88
57	Yumu	56·3	173·3	83·1	—3·5	99·4	38	1·65	1·69	114/67
66	Pintobe	54·2	171·4	80·4	—5·3	101·4	33	—	—	108/74
67	,,	52·0	166·0	81·5	—1·5	98·5	30	1·56	1·52	108/66
70	,,	48·6	159·3	77·4	—2·2	101·6	40	1·48	1·40	105/74
72	,,	43·5	158·3	76·1	—3·0	99·5	16	1·4	1·38	110/78

The tidal air in some instances ran parallel with the skin temperature rise, but no obvious correlation exists; the respiratory rate and pulse rate similarly did not run parallel, but it is clear that skin circulation makes demands on cardiac output, and that heat loss is favoured by pulmonary ventilation as in No. 5, 12/8/32. In only one instance did the black velvet temperature exceed that of the skin, vide No. 67, 19/8/32, and that was on a day with low cooling power despite low relative humidity. The surface area measurements, using the linear formula of Du Bois, give results of the same order as those obtained from the application of the height/weight formula, but the pelidisi, with three exceptions, are lower than Von Pirquet's standard (1915), though of the same order as those found by Benedict for white Americans. Attention must be drawn to the fact that the three exceptions are not N'galias and that the leg length is greater than half the total stature in all but three cases. These measurements agree with those of the tribes north and west of Cockatoo Creek—some 300 miles to the northward

of Mount Liebig, and differ from those obtained on the coastal tribes near the head of the Australian Bight.

DISCUSSION.

From results of oxygen consumption measurements in the field, it appears that at both Cockatoo Creek and Mount Liebig, in tropical Central Australia, where low night temperatures alternate with high day temperatures in the winter, the heat loss, which might be anticipated to raise the oxygen usage, does not in fact do so. Body temperatures and skin temperatures are both low, and the torpor of the natives described as occurring in the extreme cold of Cockatoo Creek would appear to be more appropriate to the corrected values than those previously published (1933). There is no waste of energy within the limits of error of the method used, i.e. ± 10 p.c., and it would appear that heat is conserved by effective vaso-constriction, while the radiation from small camp-fires placed close to the body serves to reduce the temperature gradient effectively. The fires, sometimes three per native, or more often three to two natives, are purposely kept small so as to require little replenishing, and so that they may be almost touching the body, burns being common during sleep. The Mount Liebig results for early morning corroborated those obtained at Cockatoo Creek, except that temperatures were not so low, but katathermometer readings showed a high cooling power for the atmosphere.

The outstanding result of the gaseous exchange measurements is the disclosure of a rapid and extensive specific dynamic response to the ingestion of almost raw meat, and the extensive change in respiratory quotient during the experimental period. The former may be associated with the fact that the meat was raw and the various extractives unchanged either in nature or quantity as compared with the protein in Benedict and Carpenter's experiments. Nevertheless, it is clear that no defect in the specific dynamic response is present. The apparently unstable respiratory quotient, which was also entirely unexpected, may be the result of border-line nutrition. The nature of the country is such that it must be relatively rare for these tribes to experience plenty, and more often they must be merely subsisting. It is quite possible that such environmental experience is responsible for this instability in the respiratory quotient.

The fall in oxygen consumption with rising temperature is to be expected, but it is not of the low order of the average summer values obtained at Koonibba, where —28 p.c. was the average deficiency. The cold nights may, however, be responsible for a higher general usage of oxygen in the winter months. To secure evidence, therefore, of a low metabolism in naked natives like these, the criteria require definition. Are summer results only to be taken? The post-absorptive, warm bed conditions of a nutrition laboratory are obviously not applicable to a primitive race. The specific dynamic response, it was hoped, would assist in coming to a conclusion, but it has failed to show defective response, which might

have been expected were low metabolism rate a fundamental feature of the aboriginal reaction to his environment. The surface area and pelidisi values closely approximate those of Western Europeans. In this the Cockatoo Creek results are entirely corroborated, and the high pelidisi values at Koonibba for the Kokata tribe point to an essential difference in body form—i.e. a shorter leg length—which may have some anthropological significance.

SUMMARY.

1. Further oxygen-consumption estimations on naked Central Australian aboriginals in a state of nature disclose no essential departure from the normal, even in the cold of the early morning.

2. Corrected values for previously published results substantiate this conclusion.

3. The specific dynamic response to ingestion of raw meat protein is both rapid and extensive, rising in some cases to + 80 p.c. in five hours.

4. The respiratory quotient after protein ingestion rises rapidly towards unity in five hours, and in the fasting subject falls towards 0·7 in the same period.

5. Surface area measurements and pelidisi values are similar to those for western European races, and differ from those obtained on coastal tribes near the head of the Australian Bight.

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